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☐ 1. Document ID: US 5997865 A

L5: Entry 1 of 3 File: USPT Dec 7, 1999

US-PAT-NO: 5997865
DOCUMENT-IDENTIFIER: US 5997865 A

TITLE: Agonist antibodies against the flk2/flt3 receptor and uses thereof

DATE-ISSUED: December 7, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bennett; Brian D.	South San Francisco	CA	94080	N/A
Broz; Susan D.	South San Francisco	CA	94080	N/A
Matthews; William	South San Francisco	CA	94080	N/A
Zeigler; Francis C.	South San Francisco	CA	94080	N/A

US-CL-CURRENT: 424/130.1; 424/143.1, 530/387.3, 530/388.22, 530/389.1

ABSTRACT:

Agonist antibodies are disclosed which bind to the extracellular domain of the flk2/flt3 receptor and thereby activate the intracellular kinase domain thereof. The labeled antibodies are useful as diagnostics for detecting the presence of the flk2/flt3 receptor in primitive hematopoietic cells for example. The antibodies are able to cause primitive hematopoietic cells to proliferate and/or differentiate and thereby enhance repopulation of mature blood cell lineages in a mammal which has undergone chemo- or radiation therapy or bone marrow transplantation. The antibodies are further useful for treating mammals which have suffered a decrease in blood cells as a consequence of disease or a hemorrhage, for example.

20 Claims, 12 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Desc | Image

☐ 2. Document ID: US 5877396 A

L5: Entry 2 of 3 File: USPT Mar 2, 1999

AC133 antibody
Stem cell surface

US-PAT-NO: 5877396
DOCUMENT-IDENTIFIER: US 5877396 A

TITLE: Mice mutant for functional Fc receptors and method of treating autoimmune diseases

DATE-ISSUED: March 2, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Ravetch; Jeffrey V.	New York	NY	N/A	N/A
Takai; Toshiyuki	Okayama	N/A	N/A	JPX
Sylvestre; Diana	New York	NY	N/A	N/A
Clynes; Raphael	New York	NY	N/A	N/A

US-CL-CURRENT: 800/3; 424/9.1, 424/9.2, 800/11, 800/18, 800/9

ABSTRACT:

Disclosed herein is a non-naturally occurring non-human vertebrate animal incapable of expressing a functional Fc receptor which may optionally be capable of expressing a protein which comprises a domain of a human Fc receptor, as well as DNA encoding such Fc receptor-based proteins. Also disclosed are in vivo methods for identifying proinflammatory agents that depend on a functional Fc receptor, in vivo methods for identifying proinflammatory agents that do not depend on a functional Fc receptor, and both in vivo and in vitro methods of identifying anti-inflammatory agents. Pharmaceutical compositions containing, and methods of treating inflammation with anti-inflammatory agents are also described.

18 Claims, 112 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 48

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw. Desc | Image

☐ 3. Document ID: US 5635388 A

L5: Entry 3 of 3 File: USPT Jun 3, 1997

US-PAT-NO: 5635388
DOCUMENT-IDENTIFIER: US 5635388 A

TITLE: Agonist antibodies against the flk2/flt3 receptor and uses thereof

DATE-ISSUED: June 3, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bennett; Brian D.	Pacifica	CA	N/A	N/A
Broz; Susan D.	San Bruno	CA	N/A	N/A
Matthews; William	Woodside	CA	N/A	N/A
Zeigler; Francis C.	San Mateo	CA	N/A	N/A

US-CL-CURRENT: 435/334; 424/85.1, 424/85.2, 424/85.5, 435/320.1, 435/328, 435/70.21, 530/351, 530/387.3, 530/388.22, 530/389.1, 536/23.53

ABSTRACT:

Agonist antibodies are disclosed which bind to the extracellular domain of the flk2/flt3 receptor and thereby activate the intracellular kinase domain thereof. The labeled antibodies are useful as diagnostics for detecting the presence of the flk2/flt3 receptor in primitive hematopoietic cells for example. The antibodies are able to cause primitive hematopoietic cells to proliferate and/or differentiate and thereby enhance repopulation of mature blood cell lineages in a mammal which has undergone chemo- or radiation therapy or bone marrow transplantation. The antibodies are further useful for treating mammals which have suffered a decrease in blood cells as a consequence of disease or a hemorrhage, for example.

15 Claims, 12 Drawing figures Exemplary Claim Number: 1
Number of Drawing Sheets: 4

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FILE 'HCAPLUS' ENTERED AT 08:45:19 ON 28 JUN 2000

L1 3528 S STEM(10A)CELL(10A)FACTOR
L2 3036 S STEM(5A)CELL(5A)FACTOR
L3 492 S L1 NOT L2
L4 21434 S SCF
L5 1255 S L4 AND STEM(L)CELL(L)FACTOR
L6 3049 S L2,L5
L7 7 S L6 AND DERMATITIS
L8 101 S L6 AND SKIN
L9 25 S L6 AND SKIN(L)(DISEASE OR DISORDER)
L10 28 S L8 AND ?PIGMENT?
L11 30 S L6 AND MASTOCYT?
L12 4 S L6 AND URTICAR?
L13 452 S L6 AND MAST (L) CELL
L14 56 S L8 AND L13
L15 19 S L8 AND ?INFLAM?
L16 6 S L6 AND (ECZEM? OR ACNE? OR PSORIA? OR ANTIECZEM? OR ANTIACNE?
L17 167 S L6 AND (MELAN? OR KERATIN?)
L18 47 S L8 AND L17
L19 322 S L6 AND (TYROSINE(L)KINASE)
L20 20 S L8 AND L19
L21 17 S L6 AND ?ASTHM?
L22 1 S L21 AND (METHOD# AND ANTIBOD?)/TI
L23 182 S L6 AND (?ALLERG? OR HYPERSENS? OR HYPOSENS? OR SENSITIV?)
L24 7 S L23 AND L8
L25 24 S L23 AND (LUNG OR PULMON? OR BROCH? OR AIRWAY OR RESPIR? OR BR
L26 2 S L25 AND (SKIN OR HYPERREAC?)/TI
L27 27 S L23 AND ?HISTAMIN?
L28 16 S L27 NOT L24-L26
L29 3 S L28 AND (NASAL OR NOSE)
L30 1 S L23 AND (?RHIN? OR ?NASAL? OR NOSE) NOT L24-L29
L31 9 S L6 AND ?ANAPHYL?
L32 5 S L31 AND (RECOMBINANT OR EFFECT#)/TI
L33 2 S L32 AND (MELANOCYT? OR SURVIVAL)/TI
L34 7 S L6 AND SHOCK
L35 0 S L34 AND L23
L36 0 S L34 AND L31
L37 785 S L6 AND (C(A)KIT OR CKIT)
L38 175 S L37 AND ?SIGNAL?
L39 112 S L37 AND STROMA?
L40 8 S L39 AND (?GASTRO? OR ?GASTRI? OR ?INTESTIN? OR DIGEST?)
L41 3 S L40 AND GAIN/TI
L42 59 S L37 AND GERM (L) CELL
L43 13 S L42 AND (?TUMOR? OR ?NEOPLAS? OR ?CANCER? OR ?CARCIN? OR ?MET
L44 8 S L43 NOT (ANEMIA OR STEEL FACTOR OR BUFFALO OR THYMOCYTES)/TI
L45 41 S L42 AND GERM CELL NOT L43
L46 1 S L45 AND CLINICAL
L47 8 S L37 AND ACK2
L48 33 S L37 AND ?DIMER?
L49 11 S L6 AND ?BRONCH?
L50 1 S L49 AND RECOMBIN?/TI
L51 124 S L6 AND (CONTRACEP? OR PREGNAN? OR FERTIL? OR INFERTIL? OR SPE
L52 25 S L22,L30,L33,L41,L44,L46,L47,L50
L53 359 S L7,L9-L12,L14-L16,L18,L20,L24-L29,L31-L34,L40-L51
L54 23 S L52 AND L53
L55 25 S L52,L54
L56 336 S L53 NOT L55
L57 17 S L56 AND (1 OR 63)/SC
L58 20 S L56 AND (1 OR 63)/SX

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L59          34 S L57,L58
L60         145 S L56 AND 15/SC,SX
L61          12 S L60 AND L59
L62          11 S L61 NOT 3/SC,SX
L63           1 S L62 AND RHINITIS
L64          22 S L59 NOT L61
L65           4 S L64 AND CYCLOSPORIN
L66           1 S L65 AND FK 506
L67          30 S L55,L63,L65,L66

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FILE LAST UPDATED: 27 Jun 2000  (20000627/ED)

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L67  ANSWER 1 OF 30  HCAPLUS  COPYRIGHT 2000 ACS
AN   2000:277810  HCAPLUS
DN   132:326056
TI   Systems for oral delivery
IN   Russell-Jones, Gregory John
PA   Biotech Australia Pty. Ltd., Australia
SO   PCT Int. Appl., 32 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
ICI  A61
CC   63-6 (Pharmaceuticals)
FAN.CNT 1
     PATENT NO.        KIND  DATE           APPLICATION NO.   DATE
     ---------------   ----  --------       ---------------   --------
PI   WO 2000022909      A2   20000427       WO 1999-IB1872    19991018
         W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU,
             CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,
             IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA,
             MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI,
             SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
             DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
             CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
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PRAI US 1998-PV104827 19981019

AB A pharmaceutical and a biol. active substance, for oral administration, can be "coated" or "encapsulated" with a carboxylic acid, such that the substance is protected from proteolysis in the stomach and is taken up from the intestine. It is thought that the carboxylic acids coat and protect the active agent from the proteolytic environment of the stomach, allowing the agent to pass safely through the stomach and to be absorbed in the small intestines. The carboxylic acid agent complex can be adopted for oral, **nasal**, buccal, and transdermal delivery of moderately sol. and even insol. bioactive agents.

ST carboxylate enteric coating encapsulation

IT Eotaxin
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(2; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Platelet-derived growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(AA; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Platelet-derived growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(AB; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Platelet-derived growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(BB; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Chemokines
(C-X-C, SDF-1/PBSF; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Chemokines
(C-X-C, SDF-1.alpha./PBSF; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Chemokines
(C-X-C, SDF-1.beta./PBSF; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Chemokines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(ENA 78; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Hemopoietins
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Flt-3 ligand; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Immunostimulants
(adjuvants; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Drug delivery systems
(aerosols; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Diagnosis
(agents; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Lipids, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(blood, regulators; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Neurotrophic factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(brain-derived; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Drug delivery systems
(capsules; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Adrenoceptor agonists
Allergy inhibitors

Analgesics
Anthelmintics
Anti-inflammatory agents
Antiarrhythmics
Antibiotics
Anticoagulants
Anticonvulsants
Antidepressants
Antidiabetic agents
Antihistamines
Antihypertensives
Antiparkinsonian agents
Antipsychotics
Antitumor agents
Antitussives
Antiviral agents
Anxiolytics
Appetite depressants
Blood products
Cholinergic agonists
Diuretics
Dopamine agonists
Expectorants
Fungicides
Hemostatics
Hypnotics and Sedatives
Imaging agents
Immunosuppressants
Inotropics
Muscarinic antagonists
Muscle relaxants
Radiopharmaceuticals
Thyroid gland
Tranquilizers
Vasodilators
Wound healing promoters
(carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Angiogenic factors
CTLA-4 (antigen)
Carboxylic acids, biological studies
Chemotactic factors
Ciliary neurotrophic factor
Corticosteroids, biological studies
Eotaxin
Erythropoietin receptors
Hepatocyte growth factor
Insulin-like growth factor receptors
Interferons
Interleukin 10
Interleukin 11
Interleukin 12
Interleukin 13
Interleukin 15
Interleukin 16
Interleukin 17
Interleukin 18
Interleukin 1.alpha.
Interleukin 1.beta.
Interleukin 2
Interleukin 3
Interleukin 4
Interleukin 5
Interleukin 6
Interleukin 7
Interleukin 8

Interleukin 9
Lactoferrins
Lymphotoxin
Macrophage inflammatory protein 1.alpha.
Macrophage inflammatory protein 1.beta.
Macrophage inflammatory protein 2
Macrophage migration inhibitory factor
Midkines
Monocyte chemoattractant protein-1
Neuropeptides
Platelet-derived growth factors
Pleiotrophins
Prostaglandins
RANTES (chemokine)
Sex hormones
Stem cell factor
Steroids, biological studies
Tumor necrosis factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Glycosides
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cardiac; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Neurotrophic factor receptors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(ciliary; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Imaging agents
(contrast, radiog.; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Imaging agents
(contrast; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Neurotrophic factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(glial derived; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Proteins, specific or class
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(granulocyte chemotactic, carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Proteins, specific or class
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(latency-assocd.; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Drug delivery systems
(lotions; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Drug delivery systems
(lozenges; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Cytokines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(macrophage inflammatory protein 3.beta.; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Chemokines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(macrophage-derived; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Proteins, specific or class
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(macrophage-stimulating; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Antibodies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(monoclonal; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Chemokines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(monocyte chemoattractant protein 3; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Cytokines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(monocyte chemoattractant protein 4; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Drug delivery systems
(ointments, creams; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Drug delivery systems
(ointments; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Growth factors, animal
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**placenta**; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Drug delivery systems
(powders; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Drug delivery systems
(suppositories; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Drug delivery systems
(syrups; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Drug delivery systems
(tablets; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Drug delivery systems
(tinctures; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Transforming growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.alpha.-; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Heregulins
Interferons
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.alpha.; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Adrenoceptor antagonists
(.beta.-; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Transforming growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.beta.-; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Transforming growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.beta.1-; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Transforming growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.beta.2-; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Transforming growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.beta.3-; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)
IT Microglobulins

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.beta.2-; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Transforming growth factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.beta.5; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT Interferons
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.gamma.; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT 50-02-2 50-33-9, Phenylbutazone, biological studies 50-56-6, Oxytocin, biological studies 53-86-1, Indomethacin 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 60-33-3, Linoleic acid, biological studies 76-93-7, Benzilic acid, biological studies 83-49-8, Hyodeoxycholic acid 85-01-8, Phenanthrene, biological studies 91-20-3, Naphthalene, biological studies 92-13-7, Pilocarpine 92-92-2, 4-Biphenylcarboxylic acid 98-73-7, 4-tert-Butylbenzoic acid 106-14-9, 12-Hydroxystearic acid 112-37-8, Undecanoic acid 112-38-9, Undecylenic acid 112-79-8, Elaidic acid 112-80-1, Oleic acid, biological studies 123-76-2, Levulinic acid 126-07-8, Griseofulvin 127-27-5, Pimaric acid 128-13-2, Ursodeoxycholic acid 129-20-4, Oxyphenbutazone 130-15-4, 1,4-Naphthalenedione 141-22-0, Ricinoleic acid 143-07-7, Dodecanoic acid, biological studies 302-79-4, Retinoic acid 303-98-0, Ubidecarenone 334-48-5, Decanoic acid 373-49-9, Palmitoleic acid 459-67-6, Hydnocarpic acid 463-40-1, Linolenic acid 474-25-9, Chenodeoxycholic acid 503-07-1, Vernolic acid 506-25-2, Isanic acid 506-26-3, .gamma.-Linolenic acid 506-30-9, Eicosanoic acid 506-32-1, Arachidonic acid 514-10-3, Abietic acid 524-42-5, 1,2-Naphthalenedione 525-66-6, Propranolol 530-78-9, Flufenamic acid 544-63-8, Tetradecanoic acid, biological studies 544-64-9, Myristoleic acid 611-95-0, 4-Benzoylbenzoic acid 621-82-9, Cinnamic acid, biological studies 641-81-6, Apocholic acid 646-30-0, Nonadecanoic acid 693-72-1, Vaccenic acid 1142-39-8, 4-Hexyloxybenzoic acid 1406-18-4, Vitamin E 2168-75-4, Ethyl 3,5-diacetamido-2,4,6-triiodobenzoate 2270-20-4, 5-Phenylvaleric acid 2430-94-6, cis-5-Dodecenoic acid 2493-84-7 2608-24-4, Piposulfan 2777-65-3, 10-Undecynoic acid 2984-55-6, 2-Hydroxydodecanoic acid 3115-49-9, (p-Nonylphenoxy)acetic acid 3575-31-3, 4-Octylbenzoic acid 4419-39-0, Beclomethasone 4521-28-2, 4-(4-Methoxyphenyl)-butyric acid 5104-49-4, Flurbiprofen 5451-55-8, 4-tert-Butylcyclohexanecarboxylic acid 5728-52-9, 4-Biphenylacetic acid 5731-13-5 6402-36-4, Traumatic acid 6950-82-9, 7-Hydroxycoumarin-4-acetic acid 6990-06-3, Fusidic acid 7689-03-4, Camptothecin 8001-27-2, Hirudin 9001-12-1, MMP-1 9001-27-8, Factor VIII 9001-28-9, Factor IX 9002-64-6, Parathyroid hormone 9003-99-0, Myeloperoxidase 9004-10-8, Insulin, biological studies 9005-49-6, Heparin, biological studies 9007-12-9, Calcitonin 9014-00-0, Luciferase 9014-42-0, Thrombopoietin 9034-40-6D, LHRH, analogs 9041-92-3 9054-89-1, Superoxide dismutase 9061-61-4, Nerve growth factor 11000-17-2, Vasopressin 11096-26-7, Erythropoietin 13539-59-8, Azapropazone 13598-36-2D, Phosphonic acid, alkylidenebis-derivs. 15307-86-5, Diclofenac 15687-27-1, Ibuprofen 15872-42-1, 4-Heptyloxybenzoic acid 15872-43-2, 4-Nonyloxybenzoic acid 15872-44-3, 4-Undecyloxybenzoic acid 17230-88-5, Danazol 20651-71-2, 4-Butylbenzoic acid 21643-38-9, 4-Hexylbenzoic acid 22071-15-4, Ketoprofen 22204-53-1, Naproxen 23812-34-2 25167-62-8, Docosahexaenoic acid 25354-97-6, 2-Hexyldecanoic acid 25378-27-2, Eicosapentaenoic acid 26171-23-3, Tolmetin 26764-41-0, Eicosenoic acid 27070-56-0, Eicosatrienoic acid 29679-58-1, Fenoprofen 29973-91-9, 4-Benzyloxy-3-methoxyphenylacetic acid 30748-29-9, Feprazone 34645-84-6, Fenclofenac 36322-90-4, Piroxicam 38194-50-2, Sulindac 38289-29-1, trans-4-Pentylcyclohexanecarboxylic acid 38350-87-7, 4-Heptylbenzoic acid 51110-01-1, Somatostatin 53483-12-8 55837-18-8, Butibufen 58574-03-1, 4'-Hydroxy-4-biphenylcarboxylic acid 58957-92-9, Idarubicin 59865-13-3, **Cyclosporin** 62229-50-9, Epidermal

growth factor 67763-96-6, Insulin-like growth factor I 67763-97-7, Insulin-like growth factor II 74397-12-9, Limaprost 79955-99-0, MMP-3 81627-83-0, Macrophage colony stimulating factor 83869-56-1, Granulocyte macrophage colony stimulating factor 85637-73-6, Atriopeptin 105844-41-5, Plasminogen activator inhibitor 106096-92-8, Endothelial cell growth factors 106096-93-9, Fibroblast growth factor basic 106956-32-5, Oncostatin M 107000-34-0 113427-24-0 117147-70-3, Amphiregulin 120373-36-6, Unoprostone 121181-53-1, Filgrastim 122312-54-3, Epoetin beta 122320-05-2, Secretoryleukocyte protease inhibitor 123584-45-2, Fibroblast growth factor 4 123626-67-5, Endothelin-1 123774-72-1, Sargramostim 127464-60-2, Vascular endothelial growth factor 129653-64-1, Fibroblast growth factor 5 130939-41-2, Fibroblast growth factor 6 130939-66-1, Neurotrophin 3 139639-23-9, Tissue plasminogen activator 141256-52-2, MMP 7 143011-72-7, Granulocyte colony stimulating factor 143090-92-0, Anakinra 143375-33-1, Neurotrophin 4 146480-35-5, MMP 2 146480-36-6, MMP 9 148348-15-6, Fibroblast growth factor 7 151185-16-9, Fibroblast growth factor 9 155646-83-6, Heregulin-.beta.1 163150-12-7, Betacellulin 169494-85-3, Leptin 169592-56-7, Apopain 214210-48-7, **Placenta** growth factor 2 265112-35-4
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT 9004-06-2, Elastase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitor; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

IT 164003-41-2, Fibroblast growth factor 8
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(isoforms b and c; carboxylic acids for encapsulating or enteric coating biol. active agents for delivery to intestine)

L67 ANSWER 2 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 2000:162430 HCAPLUS
TI **Stem cell factor** is not essential for **cell** survival and proliferation of soft tissue sarcoma of neuroectodermal origin
AU Ricotti, Emanuela; Bertorello, Nicoletta; Vai, Sergio; Pagani, Alberto; Di Montezemolo, Luca Cordero; Madon, Enrico; Basso, Giuseppe
CS Department of Pediatrics, University of Turin, Turin, 10131, Italy
SO Haematologica (1999), 84(10), 879-886
CODEN: HAEMAX; ISSN: 0390-6078
PB Ferrata Storti Foundation
DT Journal
LA English
CC 15 (Immunochemistry)
AB Background and Objectives. **Stem cell factor** (**SCF**), and its receptor (**c-kit**) play key roles in the expansion and differentiation of hematopoietic progenitor **cells**, melanoblasts and primordial **germ cells**, making it possible that **SCF** and **c-kit** are involved in **neoplastic** processes deriving from these **cells**. **C-kit** has been described to be expressed at different levels in neuroblastoma and in soft tissue sarcoma of neuroectodermal origin, and seems to be required for survival processes. In this study we investigate how **c-kit** expression is regulated and whether a **SCF** autocrine loop is essential for survival of sarcoma **cell** lines. Design and Methods. **C-kit** modulation and internalization was evaluated incubating **cells** with rhSCF. **Cell** differentiation and proliferation expts. were performed to test whether **c-kit** expression is related to **cell** cycle progression or to differentiation processes. **Cell** cultures were treated with neutralizing antibody and antisense oligonucleotides in order to assess the possible significance of the **SCF** autocrine loop. Results. In vitro **SCF** stimulation induces **c-**

kit down-regulation; this phenomenon could be connected with receptor internalization, and new protein synthesis is necessary for its re-expression. The **cell** proliferation arrest in G0/G1 does not modify **c-kit** expression while down-regulation of **c-kit** was demonstrated after **cells** had been treated with differentiating agents. **SCF** neutralization does not influence either the S phase or apoptosis in sarcoma **cell** lines. Interpretation and Conclusions. In sarcoma **cell** lines, **c-kit** is regulated by differentiation processes; moreover our results suggest that **c-kit** activity, but probably not the **SCF** autocrine loop, is essential for survival of these **cell** lines.

RE.CNT 23
RE
(1) Alai, M; J Biol Chem 1992, V267, P18021 HCAPLUS
(2) Beck, D; Blood 1995, V86, P3132 HCAPLUS
(3) Broudy, V; Blood 1997, V90, P1345 HCAPLUS
(4) Campana, D; J Immunol 1987, V138, P648 HCAPLUS
(5) Carroll, M; J Biol Chem 1990, V265, P19812 HCAPLUS
(6) Carroll, M; J Biol Chem 1991, V266, P14964 HCAPLUS
(7) Cavazzana, A; Am J Pathol 1987, V127, P507 MEDLINE
(8) Cohen, P; Blood 1994, V84, P3465 HCAPLUS
(9) Hirota, S; Brain Res Mol Brain Res 1992, V15, P47 HCAPLUS
(10) Horie, M; J Biol Chem 1993, V268, P968 HCAPLUS
(11) Matsumi, Y; Nature 1990, V347, P667
(12) Miyazawa, K; Blood 1994, V83, P137 HCAPLUS
(13) Miyazawa, K; Exp Hematol 1991, V19, P1110 HCAPLUS
(14) Pagani, A; Int J Cancer 1995, V63, P738 MEDLINE
(15) Ricotti, E; Blood 1998, V91, P2397 HCAPLUS
(16) Schimdt, D; Cancer 1991, V68, P2251
(17) Shimizu, Y; J Immunol 1997, V156, P3443
(18) Thiele, C; Cancer Metastasis Rev 1991, V10, P311 MEDLINE
(19) Timeus, F; Exp Hematol 1997, V25, P1253 HCAPLUS
(20) Torti, M; J Biol Chem 1992, V267, P8293 HCAPLUS
(21) Triche, T; Hum Pathol 1983, V14, P569 MEDLINE
(22) Weiler, S; Blood 1996, V87, P3688 HCAPLUS
(23) Yee, N; J Biol Chem 1994, V269, P31991 HCAPLUS

L67 ANSWER 3 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 2000:15227 HCAPLUS
DN 132:77836
TI Improved process for preparing Schiff base adducts of amines with o-hydroxy aldehydes and compositions of matter based thereon
IN Hay, Bruce Allan; Clark, Michael Thomas
PA Pfizer Products Inc., USA
SO PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07K001-107
ICS C07K014-61; A61K047-48
CC 17-6 (Food and Feed Chemistry)
Section cross-reference(s): 18, 24, 27, **63**

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000000507	A1	20000106	WO 1999-IB993	19990602
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9938424	A1	20000117	AU 1999-38424	19990602

PRAI US 1998-PV90714 19980626
US 1998-90714 19980626
WO 1999-IB993 19990602
OS MARPAT 132:77836

AB An improved process is described for prepg. Schiff base condensation adduct final products whose components comprise a protein having beneficial activity in animals, and an arom. o-hydroxy aldehyde, which comprises bringing together the above-mentioned components in an aq. environment at a pH of 7.0 or higher to form a reaction mixt., under conditions effective to drive said condensation reaction substantially to completion by removing from about 97.0 % to about 99.9 % by wt., preferably from about 98.0 % to about 99.0 % by wt. of the water already present or produced during said condensation reaction, consistent with maintaining the integrity of the condensation reactants and adduct final product, and to assure a rate of conversion to said condensation adduct final product, i.e. , with resulting yield of said condensation adduct final product of equal to or greater than about 98.5 % by wt., preferably equal to or greater than about 99.5 % by wt. based on the wt. of the reactants. Preferred arom. o-hydroxy aldehydes comprise o-vanillin; salicylaldehyde; 2,3-dihydroxybenzaldehyde; 2,6-dihydroxybenzaldehyde; 2-hydroxy-3-ethoxybenzaldehyde; or pyridoxal. A very wide range of proteins may be employed. The improved process provides yields over 90 % and substantially quant. conversion of the aldehyde and protein to the condensation adduct.

ST Schiff base protein amine arom hydroxyaldehyde; hormone arom hydroxyaldehyde Schiff base; drug peptide arom hydroxyaldehyde Schiff base; growth promoter arom hydroxyaldehyde Schiff base; feed additive protein amine arom hydroxyaldehyde Schiff base

IT Proteins, specific or class
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(ECP (eosinophil cationic protein); improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Immunoglobulins
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(G; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Pituitary hormones
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(anterior; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Antiarteriosclerotics
(antiatherosclerotics; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Aldehydes, reactions
RL: RCT (Reactant)
(arom., o-hydroxy-; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT **Skin**
(artificial; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Temperature effects, biological
(cold; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Anti-**inflammatory** agents
Antiasthmatics
Antidiabetic agents

Antihypertensives
Antitumor agents
Atomizing (spraying)
Feed additives
Immunostimulants
Immunosuppressants
Macrophage
Temperature effects, biological
(improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Cytokines
Enkephalins
Growth promoters, animal
Hemoglobins
Hemopoietins
Immunoglobulins
Interferons
Interleukin 1
Interleukin 10
Interleukin 11
Interleukin 12
Interleukin 2
Interleukin 3
Interleukin 4
Interleukin 5
Interleukin 6
Interleukin 7
Interleukin 8
Interleukin 9
Interleukins
Lymphotoxin
Myoglobins
Proteins, general, biological studies
Stem cell factor
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Schiff bases
RL: FFD (Food or feed use); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Antibodies
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(monoclonal; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Analgesics
(opioid; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Drying
(spray; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Interferons
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.alpha.-2a; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Interferons
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.alpha.-2b; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Interferons
Tumor necrosis factors
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.alpha.; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Lactoglobulins
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.beta.-; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Interferons
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.beta.1a; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Interferons
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.gamma.1b; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT Interferons
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.gamma.; improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT 50-57-7, Lypressin 53-73-6, Angiotensin amide 56-59-7, Felypressin 58-82-2, Bradykinin 113-79-1, AVP 342-10-9, Kallidin 4117-65-1, Aspartocin 5534-95-2, Pentagastrin 8068-28-8, Colistimethate 9001-28-9, Blood-coagulation factor IX 9001-63-2, Lysozyme 9002-01-1, Streptokinase 9002-60-2, ACTH, biological studies 9002-61-3, Chorionic gonadotropin 9002-62-4, Prolactin, biological studies 9002-64-6, Parathyroid hormone 9002-67-9, Luteinizing hormone 9002-68-0, FSH 9002-71-5, TSH 9002-72-6, Somatotropin 9004-10-8, Insulin, biological studies 9004-10-8D, Insulin, dalanated 9005-49-6, Heparin, biological studies 9007-12-9, Calcitonin 9007-92-5, Glucagon, biological studies 9014-42-0, Thrombopoietin 9034-39-3, Somatoliberin 9034-40-6, LHRH 9034-42-8, .beta.-MSH 9035-54-5, **Placental** lactogen 9039-53-6, Urokinase 9087-70-1, Aprotinin 11000-17-2, Vasopressin 11096-26-7, Erythropoietin 15958-92-6, 1-8-Bradykinin 16679-58-6, Desmopressin 16870-37-4, Amogastrin 16960-16-0, Cosyntropin 17650-98-5, Ceruletide 24305-27-9, Thyrotropin-releasing hormone 26305-03-3 33515-09-2, Gonadorelin 33605-67-3, Cargutocin 34765-96-3, Alsactide 35115-60-7, Teprotide 37025-55-1, Carbetocin 37213-49-3, .alpha.-MSH 37228-64-1, .beta.-Glucocerebrosidase 37332-99-3, Avoparcin 37377-93-8, .beta.-LPH 39422-22-5, .gamma.-LPH 51110-01-1, Somatostatin 53714-56-0, Leuprolide 54017-73-1, Murodermin 57773-63-4, Triptorelin 57773-65-6, Deslorelin 58569-55-4, [Met5]enkephalin 58822-25-6, 1-5-.beta.-Neoendorphin (human) 59865-13-3, **Cyclosporin** 60118-07-2, Endorphin 60173-73-1, Arfalasin 60267-61-0, Ubiquitin 60731-46-6, Elcatonin 61489-71-2, Human menopausal gonadotropin 62304-98-7, Thymalfasin 62683-29-8, Colony stimulating factor 63631-40-3, DADL 64695-06-3, Des-Arg9-[Leu8]-bradykinin 64854-64-4, FK-33824 65154-06-5, PAF 65647-03-2 65807-02-5, Goserelin 67269-08-3 67422-14-4, Proinsulin

human 67763-96-6, IGF-1 67763-97-7, IGF-2 69558-55-0, Thymopentin 69671-17-6, .alpha.-Neoendorphin 71800-36-7, 1-9-Kallidin 73168-24-8 74135-04-9, Morphiceptin 74913-18-1D, Dynorphin, derivs. 75644-90-5 76712-82-8, Histrelin 76932-56-4, Nafarelin 77752-00-2, .beta.-Neoendorphin 78123-71-4, DAMGO 79804-71-0, Corticorelin 81627-83-0, M-CSF 82030-87-3, Somatrem 83150-76-9, Octreotide 83397-56-2, PL-017 83784-18-3, Lutrelin acetate 85006-82-2, Dynorphin B 88161-22-2, Dynorphin A 88373-73-3 89383-13-1, Somidobove 90779-69-4, Atosiban 96353-48-9, Somagrebove 97048-13-0, Urofollitropin 97825-00-8, [D-Phe7]-bradykinin 99283-10-0, Molgramostim 102583-46-0, Detirelix acetate 102733-72-2, Sometripor 102744-97-8, Sometribove 103060-53-3, Daptomycin 103222-11-3, Vapreotide 103429-31-8, CTOP 105857-23-6, Alteplase 105953-59-1, Dumorelin 106282-98-8, Somalapor 110551-45-6 110881-59-9 110942-02-4, Aldesleukin 111212-85-2, Ersofermin 113189-02-9, Antihemophilic factor 113427-24-0, Epoetin alfa 114455-29-7 119693-74-2, Somenopor 120993-53-5, Desirudin 121181-53-1, Filgrastim 122302-71-0, Atriopeptin-21 122384-88-7, Amlintide 122752-15-2, Deltorphin C 122752-16-3, Deltorphin B 123774-72-1, Sargramostim 126752-39-4, Somavubove 127785-64-2, Basifungin 127984-74-1, Lanreotide acetate 128270-60-0, Bivalirudin 129566-95-6, Somfasepor 135968-09-1, Lenograstim 136105-89-0 137463-76-4, PIXY321 137487-62-8, Alvircept sudotox 138614-30-9, HOE 140 139639-23-9, Tissue plasminogen activator 142298-00-8, Emoctakin 143003-46-7, Alglucerase 143090-92-0, Anakinra 148637-05-2, Cilmostim 151126-32-8, Pramlintide 152923-56-3, Daclizumab 154248-96-1, Iroplact 154248-97-2, Imiglucerase 157238-32-9, Cetermin 165101-51-9, Becaplermin 166089-33-4, Nagrestipen 171870-23-8, Lanoteplase
RL: FFD (Food or feed use); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

IT 66-72-8, Pyridoxal 90-02-8, Salicylaldehyde, reactions 121-33-5, Vanillin 387-46-2, 2,6-Dihydroxybenzaldehyde 492-88-6, 2-Hydroxy-3-ethoxybenzaldehyde 24677-78-9, 2,3-Dihydroxybenzaldehyde
RL: RCT (Reactant)
(improved process for prepg. Schiff base adducts of peptide and protein amine groups with o-hydroxy aldehydes and compns. based thereon for food and drug use)

RE.CNT 12
RE
(1) Anon; US 4886659 A
(2) Anon; US 5633351 A
(3) Brandon, D; Journal of Immunological Methods 1985, V78(1), P87 HCAPLUS
(4) Clark; US 5198422 A 1993 HCAPLUS
(5) Dalgety UK Limited; EP 0284186 A 1988
(6) Dhont, J; Aroma Res Proc Int Symp 1975, P193 HCAPLUS
(7) Dzhagarov, B; Zh Prikl Spektrosk 1994, V61(1-2), P95 HCAPLUS
(8) Neorx Corporation; WO 9003401 A 1990
(9) Tomlinson, A; Food Chemistry 1993, V48(4), P373 HCAPLUS
(10) Williams, J; Biochim Biophys Acta 1968, V154(2), P323 HCAPLUS
(11) Zaugg, R; Journal of Biological Chemistry 1977, V252(23), P8542 HCAPLUS
(12) Zhu, T; Bioconjugate Chemistry 1994, V5(4), P312 HCAPLUS

L67 ANSWER 4 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1999:780242 HCAPLUS
DN 132:217164
TI **Stem cell factor/c-kit** system in **spermatogenesis**
AU Mauduit, Claire; Hamamah, Samir; Benahmed, Mohamed
CS INSERM U407, INSERM U407, Faculte de Medecine Lyon-Sud, Oullins, F-69921, Fr.
SO Hum. Reprod. Update (1999), 5(5), 535-545
CODEN: HRUPF8; ISSN: 1355-4786
PB Oxford University Press

DT Journal; General Review
LA English
CC 2-0 (Mammalian Hormones)
Section cross-reference(s): 14
AB A review, with 92 refs., reporting a large no. of data, obtained essentially in animal models, that suggest an important role for the **SCF/c-kit** system in **spermatogenesis** and, as a corollary, its potential involvement in **spermatogenic** defects. One of the major unresolved questions with male **infertility** is the identification of the mol. origin of a great majority of the **spermatogenetic** arrests currently diagnosed as idiopathic male **infertility**. During the past years, several families of regulating **factors** have been implicated in **spermatogenesis** defects obsd. essentially in animal models. Among these **factors** are signalling mols., and particularly the **stem cell factor** (**SCF**)/**c-kit** system. The **SCF** and its receptor **c-kit** are an appropriate example to illustrate the role of signalling mols. in the physiol. and pathol. of **spermatogenesis**. The **SCF/c-kit** regulates primordial **germ cell** migration, proliferation and apoptosis during fetal gonadal development. The **SCF/c-kit** also regulates **spermatogonia** proliferation in the adult animal. In mutant mice, abnormalities of the **SCF/c-kit** gene expression, such as gene deletion, point mutation, alternative splicing defect, lead to different types of **spermatogenesis** alterations (e.g., decrease in primordial **germ cell** migration, decrease in **spermatogonia** proliferation). More recently, defects in **SCF/c-kit** gene expression have also been shown in human testicular dysfunctions. Indeed, a redn. in **SCF/c-kit** expression has been evidenced in oligozoospermia/azoospermia assocd. with an increase in the **germ cell** apoptosis process. In addn., **c-kit** seems to be a good marker of **seminoma** testicular **tumors**.
ST review **stem cell factor c kit** protein **spermatogenesis**; male **infertility** **stem cell factor c kit** protein review; **seminoma stem cell factor c kit** protein review
IT Embryo, animal
(fetus, development; **stem cell factor/c-kit** system in **spermatogenesis** in relation to)
IT **Fertility**
(male, disorder; **stem cell factor/c-kit** system in **spermatogenesis** in relation to)
IT Testis, **neoplasm**
(**seminoma**; **stem cell factor/c-kit** system in **spermatogenesis** in relation to)
IT **Spermatogenesis**
Testis
(**stem cell factor/c-kit** system in **spermatogenesis**)
IT **Stem cell factor**
c-Kit (protein)
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(**stem cell factor/c-kit** system in **spermatogenesis**)
IT Apoptosis
Cell migration
Cell proliferation
Signal transduction, biological

Testis, disease
(**stem cell factor/c-kit**
system in **spermatogenesis** in relation to)
RE.CNT 93
RE
(1) Albanesi, C; Development 1996, V122, P1291 HCAPLUS
(2) Allard, E; Biol Reprod 1996, V55, P185 HCAPLUS
(3) Anderson, D; Cell 1990, V63, P235 HCAPLUS
(4) Ashby, J; Environ Health Perspect 1997, V105, P164 HCAPLUS
(5) Bedell, M; Genes Dev 1995, V9, P455 HCAPLUS
(6) Bellve, A; J Cell Biol 1977, V74, P68 MEDLINE
(7) Benahmed, M; Male Infertility: Clinical Investigation, Cause, Evaluation and Treatment 1996, P55
(8) Blanchard, K; Endocrinology 1998, V139, P236 HCAPLUS
(9) Blume, J; EMBO J 1991, V10, P4121
(10) Bokemeyer, C; J Cancer Res Clin Oncol 1996, V122, P301 HCAPLUS
(11) Boussouar, F; Endocrinology 1999, V140, P3054 HCAPLUS
(12) Brannan, C; Genes Dev 1992, V6, P1832 HCAPLUS
(13) Brannan, C; Proc Natl Acad Sci 1991, V88, P4671 HCAPLUS
(14) Breyer, P; Endocrinology 1996, V137, P2159 HCAPLUS
(15) Carson, W; Proc Natl Acad Sci 1994, V91, P7553 HCAPLUS
(16) Chabot, B; Nature 1988, V335, P88 HCAPLUS
(17) Cheek, A; J Androl 1998, V19, P5 HCAPLUS
(18) Cooper, R; J Endocrinol 1997, V152, P159 HCAPLUS
(19) Copeland, N; Cell 1990, V63, P175 HCAPLUS
(20) DeRooj, D; Curr Opin Cell Biol 1998, V10, P694
(21) Dolci, S; Nature 1991, V352, P809 HCAPLUS
(22) Dym, M; Biol Reprod 1995, V52, P8 HCAPLUS
(23) Eddy, E; Gamete Res 1981, V4, P333
(24) Feng, H; Biol Reprod 1997, V57, P194 HCAPLUS
(25) Feng, H; Fertil Steril 1999, V71, P85 MEDLINE
(26) Flanagan, J; Cell 1991, V64, P1025 HCAPLUS
(27) Fritz, I; The Sertoli Cell 1993, P217
(28) Fusigawa, M; Urology 1998, V3, P460
(29) Geissler, E; Cell 1988, V55, P185 HCAPLUS
(30) Geissler, E; Genetics 1981, V97, P337 MEDLINE
(31) Giebel, L; Oncogene 1992, V7, P2207 HCAPLUS
(32) Giebel, L; Proc Natl Acad Sci 1991, V88, P8696 HCAPLUS
(33) Gilbert, S; Developmental Biology 1994, P788
(34) Gnessi, L; Endocr Rev 1997, V18, P541 HCAPLUS
(35) Godin, I; Nature 1991, V352, P807 HCAPLUS
(36) Hakovirta, H; Endocrinology 1999, V140, P1492 HCAPLUS
(37) Huang, E; Mol Biol Cell 1992, V3, P349 HCAPLUS
(38) Izquierdo, M; J Pathol 1995, V177, P253 MEDLINE
(39) Jiang, C; Gene 1997, V185, P285 HCAPLUS
(40) Kierszenbaum, A; Endocr Rev 1994, V15, P116 MEDLINE
(41) Lamb, D; J Androl 1999, V20, P23 MEDLINE
(42) Lev, S; J Biol Chem 1992, V267, P15970 HCAPLUS
(43) Longley, B; Proc Natl Acad Sci 1997, V94, P9017 HCAPLUS
(44) Loveland, K; J Endocrinol 1997, V153, P337 HCAPLUS
(45) Lyon, M; Genet Res 1984, V44, P161 MEDLINE
(46) Majumdar, M; J Biol Chem 1994, V269, P1237 HCAPLUS
(47) Manova, K; Dev Biol 1991, V146, P312 HCAPLUS
(48) Manova, K; Dev Biol 1993, V157, P85 HCAPLUS
(49) Manova, K; Development 1990, V110, P1057 HCAPLUS
(50) Martin, F; Cell 1990, V63, P203 HCAPLUS
(51) Marziali, G; Dev Biol 1993, V157, P182 HCAPLUS
(52) Matsui, Y; Nature 1990, V347, P667 HCAPLUS
(53) Matsui, Y; Nature 1991, V353, P750 MEDLINE
(54) Mauduit, C; J Biol Chem 1999, V274, P770 HCAPLUS
(55) Mauduit, C; Male Sterility for Motility Disorders: Etiological Factors and Treatment 1999, P173
(56) McCoshen, J; Experientia 1975, V31, P589 MEDLINE
(57) Meyts, E; J Pathol 1996, V178, P166 MEDLINE
(58) Mintz, B; J Exp Zool 1957, V134, P207
(59) Monk, M; J Embryol Exp Morphol 1981, V63, P75 MEDLINE

(60) Morrison, G; Bio Essays 1993, V15, P77
(61) Morrison, S; Cell 1997, V88, P287 HCAPLUS
(62) Motro, B; Development 1991, V102, P1207
(63) Nehar, D; Endocrinology 1997, V138, P1964 HCAPLUS
(64) Nocka, K; EMBO J 1990, V9, P1805 HCAPLUS
(65) Ogawa, M; J Exp Med 1991, V174, P63 HCAPLUS
(66) Okabe, M; Am J Hum Genet 1998, V62, P1274 HCAPLUS
(67) Orth, J; Mol Reprod Dev 1996, V45, P123 HCAPLUS
(68) Pandiella, A; J Biol Chem 1992, V267, P24028 HCAPLUS
(69) Rajpert-De Meyts, M; Int J Androl 1994, V17, P85
(70) Reith, A; EMBO J 1991, V10, P2451 HCAPLUS
(71) Reith, A; Genes Dev 1990, V4, P390 HCAPLUS
(72) Robertson, D; Molecular Biology of the Male Reproductive System 1993, P411 HCAPLUS
(73) Roosen-Runge, E; Ann NY Acad Sci 1952, V55, P574
(74) Rossi, P; Biochem Biophys Res Commun 1991, V176, P910 HCAPLUS
(75) Rossi, P; Dev Biol 1993, V155, P68 HCAPLUS
(76) Sandlow, J; J Androl 1996, V17, P403 HCAPLUS
(77) Sarvella, P; J Hered 1956, V47, P123
(78) Schlegel, P; J Androl 1999, V20, P18 MEDLINE
(79) Strohmeyer, T; J Urol 1995, V153, P511 MEDLINE
(80) Tajima, Y; Development 1991, V113, P1031 HCAPLUS
(81) Tam, P; J Embryol Exp Morphol 1981, V64, P133 MEDLINE
(82) Tan, J; Science 1990, V247, P209 HCAPLUS
(83) Tesarik, J; Mol Hum Reprod 1998, V4, P757 MEDLINE
(84) Toksoz, D; Proc Natl Acad Sci 1992, V89, P7350 HCAPLUS
(85) Vandenbark, G; Oncogene 1992, V7, P1259 HCAPLUS
(86) Verhoeven, G; J Androl 1991, V12, P9 HCAPLUS
(87) Vincent, S; Development 1998, V125, P4585 HCAPLUS
(88) Wang, R; Biol Reprod 1998, V58, P1250 HCAPLUS
(89) Wassarman, P; Reproductive Endocrinology, Surgery and Technology 1996, P341
(90) Yan, W; Endocrinology 1999, V140, P1499 HCAPLUS
(91) Yarden, Y; EMBO J 1987, V6, P3341 HCAPLUS
(92) Yee, N; J Exp Med 1994, V179, P1777 HCAPLUS
(93) Yoshinaga, K; Development 1991, V113, P689 MEDLINE

L67 ANSWER 5 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1999:776188 HCAPLUS
DN 132:135190
TI Role of the **c-Kit/SCF** system in regulation of mammalian **spermatogenesis**
AU Bartmanska, Jolanta
CS Instytut Zoologiczny, Uniwersytet Wroclawski, Pol.
SO Postepy Biol. Komorki (1999), 26(3), 461-475
CODEN: PBKODV; ISSN: 0324-833X
PB Fundacja Biologii Komorki i Biologii Molekularnej
DT Journal; General Review
LA Polish
CC 13-0 (Mammalian Biochemistry)
AB A review with 72 refs. The **c-Kit** a receptor with tyrosine kinase activity is encoded by protooncogene **c-kit** situated in the mouse W (White Spotting) locus. It is expressed on the surface of gametogenic **cells** of various developmental stage. The only **c-Kit** independent **cells** are As **spermatogonia**. **SCF** (**Stem Cell Factor**), which is a ligand for **c-Kit** receptor is encoded in the Sl (Steel) locus and expressed in Sertoli **cells**. In male gonads there are two forms of **SCF**: mbSCF (membrane bound **SCF**) and sSCF (sol. **SCF**). Both forms are able to induce receptor phosphorylation. The **c-Kit/SCF** system plays a crucial function in regulation of multiplication and migration of PGCs (Primordial **Germ Cells**) and gonocytes, and also in multiplication and surviving of **spermatogonia**. It acts to prevent **spermatocytes** and **spermatids** apoptosis. The system

serves also as a **factor** facilitating motility, capacitation and acrosomal reaction of **spermatozoa**. Mutations at the mouse W and Sl locus lead to pleiotropic effects including reduced **fertility** or sterility and severe disturbances in hematopoiesis and melanogenesis. The **c-Kit/SCF** may be involved the initiation or progression of some testis **tumors**.

ST review **cKit SCF spermatogenesis**

IT Testis
(Sertoli cell; **c-Kit/SCF** system in regulation of mammalian **spermatogenesis**)

IT **Spermatogenesis**
(**c-Kit/SCF** system in regulation of mammalian **spermatogenesis**)

IT **Stem cell factor**
c-Kit (protein)
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(**c-Kit/SCF** system in regulation of mammalian **spermatogenesis**)

IT **Fertility**
(male; **c-Kit/SCF** system in regulation of mammalian **spermatogenesis**)

IT Gamete and **Germ cell**
(primordial; **c-Kit/SCF** system in regulation of mammalian **spermatogenesis**)

IT Cell migration
(**sperm** motility; **c-Kit/SCF** system in regulation of mammalian **spermatogenesis**)

IT **Sperm**
(**spermatocyte**; **c-Kit/SCF** system in regulation of mammalian **spermatogenesis**)

IT **Sperm**
(**spermatogonium**; **c-Kit/SCF** system in regulation of mammalian **spermatogenesis**)

L67 ANSWER 6 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1999:393951 HCAPLUS
DN 131:31048
TI **Method** for treating **asthma** using **stem cell factor** (**SCF**) **antibody**
IN Brownell, Elise; Lukacs, Nicholas; Kunkel, Steven L.; Strieter, Robert M.
PA Bayer Corporation, USA; Univ. of Michigan
SO U.S., 21 pp., Cont. of U.S. Ser. No. 431,314, abandoned.
CODEN: USXXAM
DT Patent
LA English
IC ICM A61K039-395
NCL 424145100
CC 15-3 (Immunochemistry)
Section cross-reference(s): 14
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5911988	A	19990615	US 1997-912541	19970818
PRAI	US 1995-431314		19950428		

AB This invention provides pharmaceutical compns. comprising anti-**SCF** antibodies for the redn. of eosinophilia in the lungs of mammals. This invention also provides for methods of treating **asthma** and generating a murine model for **asthma**. **Asthma** model is prepd. in mice with immunization of Schistosoma mansoni egg antigen. In the invention, eosinophilia or eosinophil infiltration is also reduced by treating with anti-interleukin 4 antibodies.

ST **stem cell factor** antibody **asthma** eosinophilia; interleukin 4 monoclonal antibody eosinophil infiltration; airway inflammation **SCF** IL4 antibody; mouse model **asthma** Schistosoma egg antigen

IT **Asthma**

Disease models
Eosinophilia
(anti-**stem cell factor** antibody and anti-interleukin 4 antibody for treating **asthma** or eosinophilic airway inflammation)

IT Antibodies
RL: BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(anti-**stem cell factor** antibody and anti-interleukin 4 antibody for treating **asthma** or eosinophilic airway inflammation)

IT Interleukin 4
Stem cell factor
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(anti-**stem cell factor** antibody and anti-interleukin 4 antibody for treating **asthma** or eosinophilic airway inflammation)

IT Mouse
(**asthma** model; mice immunized with Schistosoma mansoni egg antigen for use as **asthma** model)

IT Drug delivery systems
(carriers; mice immunized with Schistosoma mansoni egg antigen for use as **asthma** model)

IT Eosinophil
(infiltration; anti-**stem cell factor** antibody and anti-interleukin 4 antibody for treating **asthma** or eosinophilic airway inflammation)

IT Respiratory tract
(inflammation, eosinophilic; anti-**stem cell factor** antibody and anti-interleukin 4 antibody for treating **asthma** or eosinophilic airway inflammation)

IT Drug delivery systems
(intra-tracheal; mice immunized with Schistosoma mansoni egg antigen for use as **asthma** model)

IT Lung
Mammal (Mammalia)
Schistosoma mansoni
(mice immunized with Schistosoma mansoni egg antigen for use as **asthma** model)

IT c-Kit (protein)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(mice immunized with Schistosoma mansoni egg antigen for use as **asthma** model)

IT Antigens
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(mice immunized with Schistosoma mansoni egg antigen for use as **asthma** model)

IT Antibodies
RL: BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; anti-**stem cell factor** antibody and anti-interleukin 4 antibody for treating **asthma** or eosinophilic airway inflammation)

IT Egg
(parasite; mice immunized with Schistosoma mansoni egg antigen for use as **asthma** model)

IT 9001-92-7, Protease
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**stem cell factor**-targeted; mice immunized with Schistosoma mansoni egg antigen for use as **asthma** model)

RE.CNT 35
RE
(1) Ackerman, V; Chest 1994, V105, P687 MEDLINE
(2) Ando, A; J Clin Invest 1993, V92, P1639 HCAPLUS

(3) Anon; WO 9217505 1992 HCAPLUS
(4) Anon; Genzyme Catalog 1993, P196
(5) Bischoff; J Exp Med 1992, V175, P237 HCAPLUS
(6) Broxmeyer, H; Cancer Cells 1991, V3(12), P480 HCAPLUS
(7) Chester; Tibtech 1995, V13, P294 HCAPLUS
(8) Dastych, J; J of Immunology 1994, V152, P213 HCAPLUS
(9) Djukanovic, R; AAS 40 Update on Childhood Asthma 1993, P169 MEDLINE
(10) Durum; Fundamental Immunology 1993
(11) Dvorak, A; Int Arch Allergy Immunology 1993, V101, P247 MEDLINE
(12) Dvorak, A; J Leukocyte Biology 1993, V54, P465 MEDLINE
(13) Galli, S; Am Jour Pathobio 1993, V142(4), P965 HCAPLUS
(14) Galli, S; J Clin Invest 1993, V91, P148 MEDLINE
(15) Godfrey, S; AAS 40 Update on Childhood Asthma 1993, P109 MEDLINE
(16) Iemura, A; Am J Pathobiology 1994, V144(2), P321 HCAPLUS
(17) Kaushansky, K; Proteins:Structure, Function, and Genetics 1992, V12, P1 HCAPLUS
(18) Kinashi, T; Blood 1994, V83(4), P1033 HCAPLUS
(19) Li, M; Nouv Rev Fr Hematologie 1993, V35, P81 MEDLINE
(20) Lukacs; European Journal of Immunology 1995, V25, P245 HCAPLUS
(21) Lukacs; Journal of Leukocyte Biology 1996, V59, P13 HCAPLUS
(22) Lukacs; The Journal of Immunology 1995, V154, P5411 HCAPLUS
(23) Lukacs; The Journal of Immunology 1996, V156, P3945 HCAPLUS
(24) Lukacs; The Journal of Immunology 1997, V158, P4398 HCAPLUS
(25) Lukacs, N; Am J Respir Mol Biol 1994, V10, P526 HCAPLUS
(26) Morley, J; Trends in Phar Sci 1993, V14, P208 MEDLINE
(27) Page, C; Am Rev Respir Dis 1993, V147, PS29 MEDLINE
(28) Rottem, M; J Immunology 1993, V151, P4950 HCAPLUS
(29) Ryan, J; J Neurosci Research 1994, V37, P415 HCAPLUS
(30) Saito, H; Int Arch Allergy Immunology 1994, V103, P143 HCAPLUS
(31) Tsuura, Y; Virchows Archiv 1994, V424, P135 MEDLINE
(32) Valent, P; Immunology Today 1994
(33) Wegner; US 5324510 1994
(34) Windt, M; Asthma:Evolving Therapeutic Regimens 1991, P23
(35) Ziegler, I; J Biol Chem 1993, V268(17), P12544 HCAPLUS

L67 ANSWER 7 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1999:327752 HCAPLUS
DN 131:128007
TI Removal of **stem cell factor** or addition of monoclonal anti-**c-KIT** antibody induces apoptosis in murine melanocyte precursors
AU Ito, Masaru; Kawa, Yoko; Ono, Hirotake; Okura, Mitsuhiro; Baba, Takako; Kubota, Yasuo; Nishikawa, Sin-Ichi; Mizoguchi, Masako
CS Department of Dermatology, St. Marianna University School of Medicine, Kawasaki, 216-8511, Japan
SO J. Invest. Dermatol. (1999), 112(5), 796-801
CODEN: JIDEAE; ISSN: 0022-202X
PB Blackwell Science, Inc.
DT Journal
LA English
CC 13-3 (Mammalian Biochemistry)
AB Previous findings indicate that the protein **c-KIT** and its ligand, **stem cell factor** (**SCF**) play a crucial role in the development of melanocytes from their precursors in the embryonic neural crest **cells**. Using a monoclonal anti-**c-KIT** antibody, **ACK2**, which is an antagonistic blocker of **c-KIT** function, we and others demonstrated that mouse melanocytes disappeared with the injection of **ACK2** during certain periods of embryonic and postnatal life. The precise mechanisms of this disappearance, however, remain unclear. Because melanocytes disappeared without any inflammation in these in vivo studies, we suspect that apoptosis was a main cause of their disappearance. In this study, to clarify the underlying mechanism, we studied whether **ACK2** induces apoptosis in **c-KIT**-pos. melanoblasts, which appear in mouse neural crest **cells** cultured with **SCF** from 9.5 d old mouse embryos.

With an in situ apoptosis detection kit, a significant increase in apoptosis was detected after the removal of **SCF**, which further increased with the addn. of **ACK2** during **SCF**-dependent periods. The occurrence of apoptosis in the cultured **cells** was also demonstrated by a DNA anal. and electron microscopy. Immunohistochem. double staining confirmed that the apoptotic **cells** were **c-KIT** pos., and the electron microscopy showed that these apoptotic **cells** were melanocyte precursors. It was therefore demonstrated that apoptosis was induced in the **SCF**-dependent **c-KIT**-pos. melanocytes in vitro when the **SCF/c-KIT** interaction was obstructed. These findings elucidate the mechanism of the regulation of melanocyte development, and the survival and proliferation of these precursor **cells**, by **SCF/c-KIT** interaction.

ST **cKit stem cell factor** apoptosis development melanocyte

IT Apoptosis
Development, mammalian postnatal
Embryo, animal
(removal of **stem cell factor** or addn. of monoclonal anti-**c-KIT** antibody induces apoptosis in murine melanocyte precursors)

IT **Stem cell factor**
c-Kit (protein)
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(removal of **stem cell factor** or addn. of monoclonal anti-**c-KIT** antibody induces apoptosis in murine melanocyte precursors)

RE.CNT 24

RE
(1) Brown, D; J Biol Chem 1993, V268, P3037 HCAPLUS
(2) Chabot, B; Nature 1988, V335, P88 HCAPLUS
(3) Cohen; Immunol Today 1993, V14, P126 HCAPLUS
(4) Gavrieli, Y; J Cell Biol 1992, V119, P493 HCAPLUS
(5) Grabbe, J; Arch Dennatol Res 1994, V287, P78 HCAPLUS
(6) Huang, E; Cell 1990, V63, P225 HCAPLUS
(7) Huang, S; Oncogene 1996, V13, P2339 HCAPLUS
(8) Iemura, A; Am J Pathol 1994, V144, P321 HCAPLUS
(9) Ito, K; J Embryol Exp Morph 1984, V84, P49 MEDLINE
(10) Kerr, J; Br J Cancer 1972, V26, P239 MEDLINE
(11) Mayer, T; Dev Biol 1973, V34, P39 MEDLINE
(12) Murphy, M; Dev Biol 1992, V153, P396 MEDLINE
(13) Nishikawa, S; EMBO J 1991, V10, P2111 HCAPLUS
(14) Ogawa, M; J Exp Med 1991, V174, P63 HCAPLUS
(15) Okura, M; J Invest Dermatol 1995, V105, P322 HCAPLUS
(16) Raff, M; Nature 1992, V356, P397 MEDLINE
(17) Raskin, C; J Am Acad Dermatol 1997, V36, P885 MEDLINE
(18) Rawles, M; Phys Zool 1947, V20, P248
(19) Schmitz, G; Anal Biochem 1991, V192, P222 HCAPLUS
(20) Steel, K; Development 1992, V115, P1111 MEDLINE
(21) Williams, D; Cell 1990, V63, P167 HCAPLUS
(22) Williams, G; Cell 1991, V65, P1097 HCAPLUS
(23) Wyllie, A; Int Rev Cytol 1980, V68, P251 MEDLINE
(24) Zsebo, K; Cell 1990, V63, P195 HCAPLUS

L67 ANSWER 8 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1999:305471 HCAPLUS
DN 131:67841
TI Effect of anti-**allergic** drugs on **histamine** release from mast cells- Analysis with cord blood-derived human cultured mast cells
AU Kanbe, Naotomo; Kurosawa, Motohiro; Igarashi, Yasushi; Amano, Hiroo; Matsushima, Youichiro; Miyachi, Yoshiki
CS Department of Dermatology, Gunma University School of Medicine, Japan

SO Ensho (1999), 19(2), 93-98
CODEN: ENSHEE; ISSN: 0389-4290
PB Nippon Ensho Gakkai Jimukyoku
DT Journal
LA Japanese
CC **1-7** (Pharmacology)
AB Mast cells have been regarded as one of the most important effector cells in IgE-dependent **allergic** response. Recently the heterogeneity of mast cells in localization and species have been recognized. However, whether anti-**allergic** drugs possess inhibitory effects on **histamine** release from human mast cells still remains uncertain. Therefore, in the present study, effects of anti-**allergic** drugs on **histamine** release from human mast cells, which were derived by the culture of cord blood cells with 80 ng/mL recombinant human **stem cell factor** and 50 ng/mL interleukin 6.
The human cultured mast cells presented functional IgE receptors on their cell surfaces and were effectively stimulated to release **histamine** in dose-dependent and time-dependent manners of anti-IgE antibody. Anti-**allergic** drugs, such as azelastine, ketotifen, and emedastine, were able to inhibit **histamine** release from the human mast cells in dose-dependent manners. The immunosuppressive agent, **cyclosporin** A, and a flavonoid, quercetin, also showed inhibitory effects on the **histamine** release from the human cultured mast cells.
ST **antiallergic histamine** mast cell IgE receptor
IT Immunoglobulins
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(E; effect of anti-**allergic** drugs on **histamine** release from mast cells- anal. with cord blood-derived human cultured mast cells)
IT Immunoglobulin receptors
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(IgE; effect of anti-**allergic** drugs on **histamine** release from mast cells- anal. with cord blood-derived human cultured mast cells)
IT **Allergy** inhibitors
Mast cell
(effect of anti-**allergic** drugs on **histamine** release from mast cells- anal. with cord blood-derived human cultured mast cells)
IT 117-39-5, Quercetin 34580-13-7, Ketotifen 58581-89-8, Azelastine 59865-13-3, **Cyclosporin** A 87233-61-2, Emedastine
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(effect of anti-**allergic** drugs on **histamine** release from mast cells- anal. with cord blood-derived human cultured mast cells)
IT 51-45-6, **Histamine**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(effect of anti-**allergic** drugs on **histamine** release from mast cells- anal. with cord blood-derived human cultured mast cells)

L67 ANSWER 9 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1999:11267 HCAPLUS
DN 130:151533
TI Stage-specific expression of the Kit receptor and its ligand (KL) during male gametogenesis in the mouse: a Kit-KL interaction critical for meiosis
AU Vincent, Stephane; Segretain, Dominique; Nishikawa, Satomi; Nishikawa, Shin-Ichi; Sage, Julien; Cuzin, Francois; Rassoulzadegan, Minoo
CS Unite 470 de l'INSERM, Faculte des Sciences, Universite de Nice, Fr.
SO Development (Cambridge, U. K.) (1998), 125(22), 4585-4593
CODEN: DEVPED; ISSN: 0950-1991
PB Company of Biologists Ltd.
DT Journal

LA English
CC 13-6 (Mammalian Biochemistry)
AB The Kit receptor and its ligand KL, which together constitute an essential effector at various stages of embryonic development, are both present during adult gametogenesis. In the testis, KL is expressed in Sertoli **cells**, and Kit in **germ cells**, starting at the premeiotic stages. A series of observations indicated previously a role in **spermatogonia** survival, without excluding a possible function at later stages. We identified a complex pattern of expression of the two components in the adult murine testis, suggestive of a role in the meiotic progression of **spermatocytes**. At stages VII-VIII of the cycle of the seminiferous epithelium, the time when **spermatocytes** enter meiosis, the membrane-assocd. form of KL extends on the Sertoli **cell** from the peripheral to the adluminal compartment of the tubule. We also found that the receptor is present on the surface of **germ cells** up to the pachytene stage. The availability of differentiated Sertoli **cell** lines, which express the KL protein and support part of the maturation of **germ cells** in coculture, allowed us to ask whether, in the in vitro reconstructed system, transit of **spermatocytes** through meiosis requires the Kit-KL interaction. Addn. of a blocking monoclonal antibody against the Kit receptor (**ACK2**) inhibited extensively the appearance of haploid **cells** and the expression of a haploid-phase-specific gene (Prm1). Recognition of the supporting Sertoli **cell** by **germ cells** was not affected, indicating a requirement for the activity of the receptor for either entering or completing meiosis. Involvement of the membrane-assocd. form of the ligand was suggested by the observation that addn. of the sol. form of KL was equally inhibitory.
ST Kit receptor **stem cell factor**
spermatogenesis meiosis cycle
IT Cell cycle
Meiosis
Seminiferous tubule epithelium
Sertoli cell
Spermatocyte
Spermatogenesis
(stage-specific expression and interaction of Kit receptor and its ligand during male gametogenesis in mouse)
IT **Stem cell factor**
c-Kit (protein)
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(stage-specific expression and interaction of Kit receptor and its ligand during male gametogenesis in mouse)
RE.CNT 29
RE
(1) Bellve, A; Methods in Enzymology 1993, V225, P84 HCAPLUS
(2) Besmer, P; Development 1993 Supplement 1993, P125 HCAPLUS
(3) Brannan, C; Genes Dev 1992, V6, P1832 HCAPLUS
(4) Chomczynski, P; Anal Biochem 1987, V162, P156 HCAPLUS
(5) Dirami, G; Signal Transduction in Testicular Cells 1996, P142
(6) Grandjean, V; Dev Biol 1997, V184, P165 HCAPLUS
(7) Harlow, E; J Virol 1981, V39, P861 HCAPLUS
(8) Huang, E; Mol Biol Cell 1992, V3, P349 HCAPLUS
(9) Jasin, M; Proc Natl Acad Sci USA 1992, V89, P10681 HCAPLUS
(10) Kunisada, T; Develop Growth Differ 1996, V38, P87
(11) Leblond, C; Ann N Y Acad Sci 1952, V55, P548
(12) Manova, K; Dev Biol 1993, V157, P85 HCAPLUS
(13) Manova, K; Development 1990, V110, P1057 HCAPLUS
(14) Meuwissen, R; EMBO J 1992, V11, P5091 HCAPLUS
(15) Motro, B; Development 1991, V113, P1207 MEDLINE
(16) Nishimune, Y; J Cell Physiol 1980, V105, P137 MEDLINE
(17) Ogawa, M; J Exp Med 1991, V174, P63 HCAPLUS
(18) Packer, A; Mol Reprod Dev 1995, V42, P303 HCAPLUS
(19) Qiu, F; EMBO J 1988, V7, P1003 HCAPLUS

(20) Rassoulzadegan, M; Cell 1993, V75, P997 HCAPLUS
(21) Rossi, P; Biochem Biophys Res Comm 1991, V176, P910 HCAPLUS
(22) Russell, L; Histological and Histopathological Evaluation of the Testis 1990
(23) Sage, J; Genomics 1997, V44, P118 HCAPLUS
(24) Sawada, K; J Reprod Fertil 1991, V93, P287 MEDLINE
(25) Sibony, M; Biol Reprod 1994, V50, P1015 HCAPLUS
(26) Sorrentino, V; Oncogene 1991, V6, P149 HCAPLUS
(27) Tajima, Y; Development 1991, V113, P1031 HCAPLUS
(28) Tajima, Y; J Reprod Fertil 1991, V91, P441 MEDLINE
(29) Yoshinaga, K; Development 1991, V113, P689 MEDLINE

L67 ANSWER 10 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1998:803214 HCAPLUS
DN 130:195670
TI **Stem cell factor** mRNA expression and production in human **nasal** epithelial **cells**: contribution to the accumulation of mast **cells** in the **nasal** epithelium of **allergy**
AU Otsuka, Hirokuni; Kusumi, Taeko; Kanai, Shozo; Koyama, Mamoru; Kuno, Yoko; Takizawa, Ryuta
CS Allergy and Immunology Laboratory. Department of Otorhinolaryngology. Nippon Medical School, Dai 2 Hospital, Kanagawa, 211, Japan
SO J. Allergy Clin. Immunol. (1998), 102(5), 757-764
CODEN: JACIBY; ISSN: 0091-6749
PB Mosby, Inc.
DT Journal
LA English
CC **15-9** (Immunochemistry)
Section cross-reference(s): **1**
AB In **allergic rhinitis**, mast **cells** are increased in no. in the epithelium of the **nasal** mucosa and play an important role in the immediate response. However, the mechanism of the accumulation is not known. The purpose of this study was to det. whether the **nasal** epithelial **cells** produce **stem cell factor** (**SCF**), the mast **cell** growth and chemoattractant **factor**, and contribute mast **cell** hyperplasia in the epithelium of **allergic rhinitis**. We have characterized the cellular localization of **SCF** using immunohistochem., reverse transcribed-PCR, and ELISA; compared **SCF** prodn. of cultured epithelial **cells** between patients with **allergic rhinitis** and **nonallergic** subjects; and compared the **SCF** prodn. with the no. of mast **cells** and the **histamine** content in the **nasal** epithelial scrapings. Immunohistochem., **SCF** was identified in the **nasal** epithelium of the biopsy specimens and in cultured **nasal** epithelial **cells**. **SCF** mRNA was expressed by cultured **nasal** epithelial **cells** not only in patients with **allergy** but also in subjects with no **allergy**. However, the **SCF**/.beta.-actin mRNA ratio and **SCF** prodn. in day 7 cultured epithelial **cells** was significantly higher in **allergic** than in **nonallergic** subjects. **SCF** prodn. from **nasal** scrapings in culture was strongly correlated with the no. of mast **cells** and the **histamine** content. These findings demonstrate that **nasal** epithelial **cells** produce **SCF** and may be important in the attraction, proliferation, and activation of mast **cells** in **allergic** inflammation in the **nose**.
ST **stem cell factor nasal** epithelium mast **cell allergic rhinitis**
IT **Allergic rhinitis**
Hyperplasia
Mast **cell**
Nasal epithelium
(**stem cell factor** mRNA expression and prodn. in human **nasal** epithelial **cells** in relation

to accumulation of mast **cells** in **allergic** **rhinitis**)

IT **Stem cell factor**
RL: ADV (Adverse effect, including toxicity); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(**stem cell factor** mRNA expression and prodn. in human **nasal** epithelial **cells** in relation to accumulation of mast **cells** in **allergic** **rhinitis**)

IT 50-24-8, Prednisolone 59865-13-3, Cyclosporin a
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**stem cell factor** mRNA expression and prodn. in human **nasal** epithelial **cells** in relation to accumulation of mast **cells** in **allergic** **rhinitis**)

IT 51-45-6, **Histamine**, biological studies
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(**stem cell factor** mRNA expression and prodn. in human **nasal** epithelial **cells** in relation to accumulation of mast **cells** in **allergic** **rhinitis**)

RE.CNT 48
RE
(1) Anderson, D; Cell 1990, V63, P235 HCAPLUS
(2) Beutler, E; J Lab Clin Med 1976, V88, P328 HCAPLUS
(3) Bischoff, S; J Exp Med 1992, V175, P237 HCAPLUS
(4) Bischoff, S; J Immunol 1997, V156, P5560
(5) Calderon, M; J Allergy Clin Immunol 1997, V99, P65 MEDLINE
(6) Chomczynski, P; Anal Biochem 1987, V162, P156 HCAPLUS
(7) Columbo, M; J Immunol 1992, V149, P599 HCAPLUS
(8) Copeland, N; Cell 1990, V63, P175 HCAPLUS
(9) Costa, J; J Exp Med 1996, V183, P2681 HCAPLUS
(10) Gibson, P; Ann Rev Resp Dis 1993, V148, P80 MEDLINE
(11) Hamann, K; Br J Dermatol 1995, V133, P203 HCAPLUS
(12) Huang, E; Cell 1990, V63, P225 HCAPLUS
(13) Irani, A; Blood 1992, V80, P3009 HCAPLUS
(14) Irani, A; Immunology 1995, V84, P72 HCAPLUS
(15) Irani, A; Proc Natl Acad Sci USA 1986, V83, P4464 HCAPLUS
(16) Ishizaka, T; Progr Allergy 1984, V34, P188 HCAPLUS
(17) Kageyama, Y; Eur J Immunol 1994, V24, P1122 HCAPLUS
(18) Kawabori, S; Jpn J Allergol 1993, V42, P894 MEDLINE
(19) Kenney, J; J Allergy Clin Immunol 1994, V93, P1060 HCAPLUS
(20) Kim, Y; J Allergy Clin Immunol 1997, V100, P389 HCAPLUS
(21) Lee, L; J Clin Invest 1995, V95, P900 HCAPLUS
(22) Longley, B; N Engl J Med 1993, V328, P1302
(23) Marini, M; J Allergy Clin Immunol 1992, V89, P1001 MEDLINE
(24) Mitsui, H; Proc Natl Acad Sci USA 1993, V90, P735 HCAPLUS
(25) Nilsson, G; J Immunol 1994, V153, P3717 HCAPLUS
(26) Ohtoshi, T; Am J Respir Cell Mol Biol 1991, V4, P255 HCAPLUS
(27) Okuda, M; Ann Allergy 1983, V50, P116 MEDLINE
(28) Okuda, M; Ann Allergy 1985, V54, P69 MEDLINE
(29) Otsuka, H; Clin Allergy 1986, V16, P589 MEDLINE
(30) Otsuka, H; J Allergy Clin Immunol 1985, V76, P695 MEDLINE
(31) Otsuka, H; J Allergy Clin Immunol 1995, V96, P528 HCAPLUS
(32) Otsuka, H; J Jpn Immunol Allergo Otolaryngol 1995, V13, P33
(33) Pesci, A; Am Rev Respir Dis 1993, V147, P684 MEDLINE
(34) Ruitenberg, E; Int Archs Allergy Appl Immunol 1982, V67, P233 MEDLINE
(35) Saito, H; J Immunol 1996, V157, P343 HCAPLUS
(36) Schleimer, R; J Allergy Clin Immunol 1985, V76, P369 MEDLINE
(37) Schwartz, L; J Immunol 1987, V138, P2611 HCAPLUS
(38) Sperr, W; Int Arch Allergy Immunol 1993, V102, P170 HCAPLUS
(39) Stellato, C; J Immunol 1995, V155, P410 HCAPLUS
(40) Strobel, S; J Clin Pathol 1981, V34, P851 MEDLINE
(41) Tsai, M; J Exp Med 1991, V74, P125

(42) Undem, B; Am J Respir Cell Mol Biol 1994, V11, P646 HCAPLUS
(43) Valent, P; Blood 1992, V80, P2237 HCAPLUS
(44) Wen, L; Biochim Biophys Acta 1996, V1314, P183 HCAPLUS
(45) Yamatodani, A; J Chromatogr 1985, V344, P115 HCAPLUS
(46) Yankaskas, J; Am Rev Respir Dis 1985, V132, P1281 MEDLINE
(47) Zsebo, K; Cell 1990, V63, P195 HCAPLUS
(48) Zsebo, K; Cell 1990, V63, P213 HCAPLUS

L67 ANSWER 11 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1998:730346 HCAPLUS
DN 130:137443
TI A novel **gain**-of-function mutation of **c-Kit** gene in **gastrointestinal stromal** tumors
AU Nakahara, Masanori; Isozaki, Koji; Hirota, Seiichi; Miyagawa, Jun-Ichiro; Hase-Sawada, Naoko; Taniguchi, Masahiko; Nishida, Toshirou; Kanayama, Suji; Kitamura, Yukihiko; Shinomura, Yasuhisa; Matsuzawa, Yuji
CS Second Department of Internal Medicine, Osaka University Medical School, Osaka, Japan
SO Gastroenterology (1998), 115(5), 1090-1095
CODEN: GASTAB; ISSN: 0016-5085
PB W. B. Saunders Co.
DT Journal
LA English
CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 3
AB The **c-Kit** gene encodes a receptor tyrosine kinase (KIT). Recently, the authors found gain-of-function mutations of the **c-Kit** gene in **gastrointestinal stromal** tumors (GISTs). All mutations were confined within the 11 amino acids (Lys-550 to Val-560) in the juxtamembrane domain, but one GIST showed a novel deletion-type mutation at codon 579 (Asp) in the juxtamembrane domain. The aim of this study was to clarify whether the mutation is activating. Mutant **c-kit** cDNA was transfected into an interleukin 3 (IL-3)-dependent Ba/F3 murine lymphoid **cell** line, and the magnitude of autophosphorylation of the mutant KIT was examd. with or without **stem cell factor** (**SCF**), a ligand of KIT. An in vitro kinase assay was also performed. The biol. behavior of the transfectant was estd. by both an in vitro proliferation assay and in vivo transplantation to nude mice. The mutant KIT exhibited constitutive phosphorylation and strong kinase activity without **SCF**. The transfectant grew autonomously without IL-3 and **SCF**, and it formed tumors in nude mice. Deletion at codon 579 (Asp) in the juxtamembrane domain of the **c-kit** gene is a novel gain-of-function mutation other than the region between Lys-550 and Val-560.
ST **cKit** gene mutation **gastrointestinal stromal** tumor
IT Autophosphorylation
Deletion (mutation)
Receptor phosphorylation
(gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)
IT **c-Kit** (protein)
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)
IT **c-kit** gene (animal)
RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(gain-of-function mutation of **c-Kit** with

constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)

IT **Stem cell factor**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)

IT **Cell** proliferation
(gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors in relation to)

IT **Digestive** system tumors
(**gastrointestinal stromal** tumor; gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)

IT Protein motifs
(juxtamembrane, mutation in; gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)

IT **Gastric** tumors
(mesenchymal stomach tumor; gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)

IT Tumors (animal)
(mesenchymal, mesenchymal stomach tumor; gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)

IT Mesenchyme
(tumors, mesenchymal stomach tumor; gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)

IT 138359-29-2
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(gain-of-function mutation of **c-Kit** with constitutive phosphorylation and kinase activity in absence of **stem cell factor** in human **gastrointestinal stromal** tumors)

RE.CNT 25
RE
(1) Besmer, P; Nature 1986, V320, P415 HCAPLUS
(2) Chabot, B; Nature 1988, V335, P88 HCAPLUS
(3) Chirgwin, J; Biochemistry 1979, V18, P5294 HCAPLUS
(4) Fishman, A; Ackerman's surgical pathology. 8th ed 1996, P645
(5) Furitsu, T; J Clin Invest 1993, V92, P1736 HCAPLUS
(6) Geissler, E; Cell 1988, V55, P185 HCAPLUS
(7) Hirota, S; Arch Pathol Lab Med 1993, V117, P996 HCAPLUS
(8) Hirota, S; Lab Invest 1995, V72, P64 HCAPLUS
(9) Hirota, S; Science 1998, V279, P577 HCAPLUS
(10) Hofstra, R; Nature 1994, V367, P375 HCAPLUS
(11) Huizinga, J; Nature 1995, V373, P347 HCAPLUS
(12) Isozaki, K; Gastroenterology 1995, V109, P456 HCAPLUS
(13) Kitamura, Y; Blood 1979, V53, P492 MEDLINE
(14) Longley, B; Nat Genet 1996, V12, P312 HCAPLUS
(15) Miettinen, M; Am J Surg Pathol 1995, V19, P207 MEDLINE
(16) Mulligan, L; Nature 1993, V363, P458 HCAPLUS

(17) Qiu, F; EMBO J 1988, V7, P1003 HCAPLUS
(18) Russell, E; Adv Genet 1979, V20, P357 HCAPLUS
(19) Stein, A; Nucleic Acids Res 1992, V20, P5237 HCAPLUS
(20) Tsujimura, T; Blood 1994, V83, P2619 HCAPLUS
(21) Tsujimura, T; Blood 1996, V87, P273 HCAPLUS
(22) Tsujimura, T; Int Arch Allergy Immunol 1995, V106, P377 HCAPLUS
(23) van de Rijn, M; Hum Pathol 1994, V25, P766 MEDLINE
(24) Williams, D; Cell 1990, V63, P167 HCAPLUS
(25) Yarden, Y; EMBO J 1987, V6, P3341 HCAPLUS

L67 ANSWER 12 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1998:414206 HCAPLUS
DN 129:188006
TI The regulation of mast **cell** development, survival and function in vivo by **stem cell factor**, the ligand for the **c-kit** receptor: **clinical** implications
AU Galli, S. J.; Costa, J. J.
CS Department of Pathology, Harvard Medical School, Beth Israel Hospital, Boston, MA, 02215, USA
SO New Trends Allergy IV Environ. Allergy Allergotoxicol. III, [Jt. Int. Symp.] (1997), Meeting Date 1995, 151-158. Editor(s): Ring, Johannes; Behrendt, Heidrun; Vieluf, Dieter. Publisher: Springer, Berlin, Germany. CODEN: 66ILAL
DT Conference; General Review
LA English
CC 15-0 (Immunochemistry)
AB A review with 41 refs. **Stem cell factor** **SCF**, the ligand for the receptor (SCFR) that is encoded by the **c-kit** protooncogene, has many important effects in mast **cell** development, survival, and function, in both humans and exptl. animals. Recombinant **SCF** (r-**SCF**) can promote mast **cell** survival by suppressing apoptosis and can induce mast **cell** hyperplasia in murine rodents, cynomolgus monkeys, baboons, and humans. R-**SCF** also can directly induce SCFR-dependent mast **cell** mediator release and can significantly modulate the extent of mast **cell** activation by Fc.epsilon.RI-dependent and certain other mechanisms. However, **SCF** can importantly influence the biol. of many **cell** types other than the mast **cell**, including hematopoietic progenitor **cells**, melanocytes and **germ cells**. Indeed, findings, in phase 1 studies of r-human **SCF** (r-hSCF) indicate that r-hSCF can promote the hyperplasia and functional activation of both mast **cells** and melanocytes. These observations have implications for the **clin.** use of r-hSCF to promote hematopoiesis, as well as for our understanding of the role of endogenous **SCF** in disorders assocd. with mast **cell** hyperplasia and/or epidermal hypermelanosis; they also point to potentially significant new therapeutic opportunities.
ST review **stem cell factor** mast **cell**
IT Hyperplasia
(mast **cell**; regulation of mast **cell** development, survival and function by **stem cell factor** and its **clin**. implications)
IT Apoptosis
Hematopoiesis
Mast **cell**
Mast **cell** activation
Melanocyte
(regulation of mast **cell** development, survival and function by **stem cell factor** and its **clin** . implications)
IT **Stem cell factor**
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(regulation of mast **cell** development, survival and function by **stem cell factor** and its **clin** . implications)

L67 ANSWER 13 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1998:390378 HCAPLUS
DN 129:135121
TI Morphological alterations in rat peritoneal mast **cells** by **stem cell factor**
AU Kim, H. M.; Shin, H. Y.; Lee, E. H.
CS Department Oriental Pharmacy, College Pharmacy, Wonkwang University, Chonbuk, S. Korea
SO Immunology (1998), 94(2), 242-246
CODEN: IMMUAM; ISSN: 0019-2805
PB Blackwell Science Ltd.
DT Journal
LA English
CC 15-9 (Immunochemistry)
Section cross-reference(s): 2
AB **Stem cell factor** (**SCF**) stimulates mast **cell** adhesion and, because **SCF** is produced normally in tissues, it may be a major **factor** responsible for the adhesion of mast **cells** to connective tissue matrix. The authors found that the morphol. of rat peritoneal mast **cells** (RPMC) altered after the addn. of recombinant murine **SCF** (rmSCF) in vitro. The ability of rmSCF to enhance morphol. alteration was dose dependent and completely abolished by anti-**c-kit** **ACK2** monoclonal antibody. Exposure of RPMC to transforming growth **factor**-.beta.1, wortmannin, genistein, herbimycin A, staurosporine, indomethacin and cytochalasin D before the addn. of rmSCF antagonized rmSCF-induced morphol. alteration. However, nordihydroguiaretic acid had no effect. Many RPMC appeared to respond also to nerve growth **factor** (NGF) but the total no. of **cells** with altered morphol. was much greater when the culture was stimulated by rmSCF than by NGF. The authors suggest that morphol. alterations of mast **cells** by rmSCF is an important step for the participation in adhesion to tissue under resident physiol. conditions.
ST morphol mast **cell stem cell factor**
IT Cytoskeleton
(in **stem cell factor** alteration of rat mast **cell** morphol.)
IT Transforming growth **factor** .beta.1
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(**stem cell factor** alteration of rat mast **cell** morphol. is inhibited by)
IT **Cell** morphology
Mast **cell**
Rat
(**stem cell factor** alters rat mast **cell** morphol.)
IT **Stem cell factor**
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(**stem cell factor** alters rat mast **cell** morphol.)
IT 80449-02-1, Tyrosine kinase 115926-52-8, Phosphatidylinositol 3-kinase 141436-78-4, Protein kinase C
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(in **stem cell factor** alteration of rat mast **cell** morphol.)
IT 506-32-1, Arachidonic acid
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(metabolites; in **stem cell factor** alteration of rat mast **cell** morphol.)
IT 9061-61-4, Nerve growth factor
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(rat mast cell morphol. is altered by)

L67 ANSWER 14 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1998:84804 HCAPLUS
DN 128:226598
TI The **c-kit** receptor and its possible signaling transduction pathway in mouse **spermatozoa**
AU Feng, Huailiang; Sandlow, Jay I.; Sandra, Alexander
CS Department of Urology, The University of Iowa, Iowa City, IA, 52242-1089, USA
SO Mol. Reprod. Dev. (1998), 49(3), 317-326
CODEN: MREDEE; ISSN: 1040-452X
PB Wiley-Liss, Inc.
DT Journal
LA English
CC 2-10 (Mammalian Hormones)
AB The presence and role of the **c-kit** protein was investigated in the mature **sperm** of the mouse. The **c-kit** monoclonal antibody (mAb) **ACK2** reacted specifically with the acrosomal region and the principal piece of fixed noncapacitated **sperm** but did not react with the acrosome region in acrosome-reacted **sperm**. **ACK2** significantly inhibited the acrosome reaction; this inhibition was relieved by the calcium ionophore A23187. The kit ligand **stem cell factor** (**SCF**) significantly increased the percentage of **sperm** undergoing acrosome reaction. This increase was partially inhibited by the calcium channel inhibitor (verapamil), the PI3k inhibitor (wortmannin), and the PLC inhibitor (U-73122). **ACK2** predominantly recognized **c-kit** proteins of 33, 48, and 150 kDa by Western blotting of mouse **sperm** exts. The 48- and 150-kDa protein bands were released into the media and tyrosine autophosphorylated at low basal levels during acrosome reaction. On stimulation with **SCF**, the level of **c-kit** phosphorylation increased significantly. These findings suggest that **c-kit** is present in mature **sperm**, and its binding to **SCF** may result in the activation of PLC.gamma.1 and PI3K, leading to receptor autophosphorylation, and ultimately may play a role in capacitation and/or the acrosome reaction.
ST **c kit** receptor acrosome signal transduction; **stem cell factor ckit** receptor acrosome
IT Acrosome
Calcium transport (biological)
Receptor phosphorylation
Signal transduction (biological)
(**c-kit** receptor and possible signaling transduction pathway in mouse **spermatozoa**)
IT **c-Kit** (protein)
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**c-kit** receptor and possible signaling transduction pathway in mouse **spermatozoa**)
IT Calcium channel
Stem cell factor
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(**c-kit** receptor and possible signaling transduction pathway in mouse **spermatozoa**)
IT 115926-52-8, Phosphatidylinositol-3 kinase
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(**c-kit** receptor and possible signaling transduction pathway in mouse **spermatozoa**)
IT 7440-70-2, Calcium, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(**c-kit** receptor and possible signaling transduction pathway in mouse **spermatozoa**)
IT 9001-86-9, Phospholipase C

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(.gamma.1; **c-kit** receptor and possible signaling transduction pathway in mouse **spermatozoa**)

L67 ANSWER 15 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1998:80821 HCAPLUS
DN 128:165683
TI **Gain**-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors
AU Hirota, Seiichi; Isozaki, Koji; Moriyama, Yasuhiro; Hashimoto, Koji; Nishida, Toshirou; Ishiguro, Shingo; Kawano, Kiyoshi; Hanada, Masato; Kurata, Akihiko; Takeda, Masashi; Muhammad Tunio, Ghulam; Matsuzawa, Yuji; Kanakura, Yuzuru; Shinomura, Yasuhisa; Kitamura, Yukihiko
CS USA
SO Science (Washington, D. C.) (1998), 279(5350), 577-580
CODEN: SCIEAS; ISSN: 0036-8075
PB American Association for the Advancement of Science
DT Journal
LA English
CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 3
AB **Gastrointestinal stromal** tumors (GISTs) are the most common mesenchymal tumors in the human **digestive** tract, but their mol. etiol. and cellular origin are unknown. Sequencing of **c-kit** complementary DNA, which encodes a proto-oncogenic receptor tyrosine kinase (KIT), from five GISTs revealed mutations in the region between the transmembrane and tyrosine kinase domains. All of the corresponding mutant KIT proteins were constitutively activated without the KIT ligand, **stem cell factor** (**SCF**). Stable transfection of the mutant **c-kit** complementary DNAs induced malignant transformation of Ba/F3 murine lymphoid **cells**, suggesting that the mutations contribute to tumor development. GISTs may originate from the interstitial **cells** of Cajal (ICCs) because the development of ICCs is dependent on the **SCF**-KIT interaction and because, like GISTs, these **cells** express both KIT and CD34.
ST **gastrointestinal stromal** tumor **ckit** mutation constitutive; gain function mutation **ckit** **gastrointestinal** tumor
IT Missense mutation
(K550I V559D; gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors)
IT **c-Kit** (protein)
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors)
IT **c-kit** gene (animal)
RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors)
IT Autophosphorylation
Receptor phosphorylation
(gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors in relation to)
IT CD34 (antigen)
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors in relation to)
IT **Stem cell factor**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(gain-of-function mutations of **c-kit** in human

gastrointestinal stromal tumors in relation to)

IT Mutation
(gain-of-function; gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors)

IT **Digestive** system tumors
(**gastrointestinal stromal** tumors (GISTs); gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors)

IT Deletion (mutation)
(in-frame, 6-bp and 15-bp and 27-bp; gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors)

IT **Intestine**
(interstitial cell of Cajal; gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors in relation to)

IT Protein motifs
(transmembrane and tyrosine kinase domains; gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors)

IT 138359-29-2
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(gain-of-function mutations of **c-kit** in human **gastrointestinal stromal** tumors)

L67 ANSWER 16 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1997:768305 HCAPLUS
DN 128:21279
TI **Stem cell factor** suppresses apoptosis in neuroblastoma **cell** lines
AU Timeus, Fabio; Crescenzio, Nicoletta; Valle, Paola; Pistamiglio, Paola; Piglione, Matilde; Garelli, Emanuela; Ricotti, Emanuela; Rocchi, Paola; Strippoli, Pierluigi; Di Montezemolo, Luca Cordero; Madon, Enrico; Ramenghi, Ugo; Basso, Giuseppe
CS Dipartimento di Scienze Pediatriche, University of Torino, Turin, 10126, Italy
SO Exp. Hematol. (Charlottesville, Va.) (1997), 25(12), 1253-1260
CODEN: EXHMA6; ISSN: 0301-472X
PB Carden Jennings Publishing
DT Journal
LA English
CC 14-1 (Mammalian Pathological Biochemistry)
AB **Stem cell factor** (**SCF**) is a glycoprotein growth **factor** produced by marrow stromal **cells** that acts after binding to its sp. surface receptor, which is the protein encoded by the protooncogene **c-kit**. **SCF** synergizes with specific lineage **factors** in promoting the proliferation of primitive hematopoietic progenitors, and has been administered to expand the pool of these progenitors in **cancer** patients treated with high-dose chemotherapy. **SCF** and its **c-kit** receptor are expressed by some **tumor cells**, including myeloid leukemia, breast **carcinoma**, small **cell** lung **carcinoma**, **melanoma**, **gynecol. tumors**, and testicular **germ cell tumors**. Previous studies of **SCF** in neuroblastoma have produced conflicting conclusions. To explore the role of **SCF** in neuroblastoma, we studied five neuroblastoma lines (IMR-S, SK-N-SH, SK-N-BE, AF8, and SJ-N-KP) and the neuroepithelioma line CHP-100. All lines expressed mRNA for **c-kit** and **c-kit** protein at low intensity as measured by flow cytometry, and secreted **SCF** in medium culture as shown by ELISA. Exogenous **SCF** did not modify 3H thymidine uptake in the neuroblastoma and neuroepithelioma **cell** lines.

After 6 days' culture in the presence of anti-**c-kit**, the no. of viable neuroblastoma **cells** was significantly lower than the control, and terminal deoxynucleotidyl transferase assay showed a substantial increase of apoptotic **cells**: The percentage of pos. **cells** was 1-3% in the control lines, whereas in the presence of anti **c-kit** it varied from 29% of SK-N-BE to 92% of CHP-100. After 9 days' culture in the presence of anti-**c-kit**, no viable **cells** were detectable. These data indicate that **SCF** is produced by some neuroblastoma **cell** lines via an autocrine loop to protect them from apoptosis.

ST neuroblastoma apoptosis **stem cell factor**

IT Apoptosis
Neuroblastoma
(**stem cell factor** suppresses apoptosis in neuroblastoma **cell** lines)

IT **Stem cell factor**
RL: BAC (Biological activity or effector, except adverse); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(**stem cell factor** suppresses apoptosis in neuroblastoma **cell** lines)

IT **c-Kit** (protein)
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**stem cell factor** suppresses apoptosis in neuroblastoma **cell** lines)

L67 ANSWER 17 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1997:669383 HCAPLUS
DN 127:314963
TI **Stem cell factor** in **nasal** polyposis and **allergic rhinitis**: increased expression by structural **cells** is suppressed by in vivo topical corticosteroids
AU Kim, Young-Ki; Nakagawa, Noriaki; Nakano, Koichi; Sulakvelidze, Irakly; Dolovich, Jerry; Denburg, Judah
CS Department of Medicine and Pediatrics, McMaster University, Hamilton, ON, L8N 3Z5, Can.
SO J. Allergy Clin. Immunol. (1997), 100(3), 389-399
CODEN: JACIBY; ISSN: 0091-6749
PB Mosby-Year Book
DT Journal
LA English
CC 2-4 (Mammalian Hormones)
AB Mast **cells** are increased in **nasal** polyp (Np) and **allergic rhinitis** (AR) tissue and are suppressed by topical corticosteroid treatment. **Stem cell factor** (**SCF**), a mast **cell** growth and survival **factor**, may explain these phenomena. We investigated structural **cell** gene expression and prodn. of **SCF** in **nasal** tissues in patients who had received and who had not received in vivo **intranasal** corticosteroid therapy. Northern blot analyses for mRNA and ELISA for biol. active **SCF** protein from cultured Np epithelial **cells** and fibroblasts were performed. Immunostaining for **SCF** in cultured and tissue **nasal** structural **cells** in the presence or absence of steroid treatment was also performed. We detected significant expression of **SCF** mRNA and protein by cultured Np epithelial **cells** and Np fibroblasts; Np fibroblast **SCF** supported the differentiation of mast **cells** in vitro. There were more immunoreactive **SCF**-pos. Np epithelial **cells** in patients with AR than in control subjects (97.2 vs. 45.6%). **SCF** that could be immunostained was significantly diminished overall in Np structural **cells** in the group given in vivo steroid treatment, with a modest (trend to significant) effect on any given **cell** type analyzed. In vitro treatment with budesonide of **SCF**-producing fibroblasts demonstrated inhibition of unstimulated, primary Np fibroblasts but not of

IL-1-stimulated fibroblasts or transformed **cell** lines. Human Np and AR tissue structural **cells** express and produce increased **SCF**. Our in vitro studies suggest that **intranasal** steroids blunt **SCF** expression in Nps, an effect that may be responsible for a decrease in mast **cells** and symptoms.

ST **stem cell factor nose** disease corticosteroid; **nasal** polyposis corticosteroid **stem cell factor**; **allergic rhinitis** corticosteroid **stem cell factor**

IT Tumors (animal)
(**nasal** polyp; topical corticosteroid suppression of increased structural **cell stem cell factor** expression in **nasal** polyposis and **allergic rhinitis**)

IT **Nose** diseases
(polyp; topical corticosteroid suppression of increased structural **cell stem cell factor** expression in **nasal** polyposis and **allergic rhinitis**)

IT **Allergic rhinitis**
Cell differentiation
Fibroblast
Gene expression
Mast **cell**
Transcription (genetic)
(topical corticosteroid suppression of increased structural **cell stem cell factor** expression in **nasal** polyposis and **allergic rhinitis**)

IT Corticosteroids, biological studies
RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(topical corticosteroid suppression of increased structural **cell stem cell factor** expression in **nasal** polyposis and **allergic rhinitis**)

IT Interleukin 1
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(topical corticosteroid suppression of increased structural **cell stem cell factor** expression in **nasal** polyposis and **allergic rhinitis**)

IT **Stem cell factor**
RL: MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(topical corticosteroid suppression of increased structural **cell stem cell factor** expression in **nasal** polyposis and **allergic rhinitis**)

IT 51333-22-3, Budesonide
RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(topical corticosteroid suppression of increased structural **cell stem cell factor** expression in **nasal** polyposis and **allergic rhinitis**)

L67 ANSWER 18 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1997:428244 HCAPLUS
DN 127:134026
TI Growth stimulation of colorectal **carcinoma** cells via the **c-kit** receptor is inhibited by TGF-.beta.1
AU Bellone, Graziella; Silvestri, Stefania; Artusio, Elisa; Tibaudi, Daniela; Turletti, Anna; Geuna, Massimo; Giachino, Claudia; Valente, Guido; Emanuelli, Giorgio; Rodeck, Ulrich
CS Department of Clinical Physiopathology, University of Torino, Turin, 10126, Italy
SO J. Cell. Physiol. (1997), 172(1), 1-11
CODEN: JCLLAX; ISSN: 0021-9541
PB Wiley-Liss
DT Journal

LA English
CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 2
AB Activation of the receptor tyrosine kinase **c-kit** by the kit-ligand, also known as **stem cell factor** (**SCF**), is essential to melanocyte and **germ cell** development and during the early stages of hematopoiesis. Deregulated expression of **c-kit** has been reported in malignancies affecting these lineages, i.e., myeloid leukemias, melanomas, and **germ cell tumors**. In addn., **c-kit** and **SCF** are coexpressed in some breast and colorectal **cancer** (CRC) **cells**, raising the question of whether **c-kit** serves an autocrine role in normal or malignant epithelial tissues. In this study, we demonstrate that human colorectal **carcinomas**, but not normal colorectal mucosa **cells**, coexpress **SCF** and **c-kit** in situ. Expression of **c-kit** was also obsd. in mucosa adjacent to colorectal **tumor** tissue. Consistent with a growth-regulatory role of **SCF** in CRC **cells**, exogenous **SCF** stimulated anchorage-dependent and anchorage-independent growth in four out of five CRC **cell** lines. Exogenous transforming growth **factor** (TGF)-.beta.1 added at nanomolar concns. to HT-29 CRC **cells**, which express the type I, II, and III TGF-.beta. receptors, downregulated **c-kit** expression to background levels and inhibited **c-kit** -dependent proliferation. Similarly, TGF-.beta.1 inhibited **SCF** -dependent proliferation of three first-passage CRC **cell** lines. In summary, expression of the potential autocrine **SCF/c-kit** axis is a **tumor**-assocd. phenomenon in colorectal **cancer** that can be suppressed by TGF-.beta.1 in TGF-.beta.-responsive CRC **cells**.
ST colorectal **carcinoma stem cell factor** TGF
IT Cell proliferation
Colorectal **carcinoma**
HT-29 cell
Proliferation inhibition
(growth stimulation of colorectal **carcinoma** cells via the **c-kit** receptor inhibition by TGF-.beta.1)
IT Transforming growth factor .beta.1
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(growth stimulation of colorectal **carcinoma** cells via the **c-kit** receptor inhibition by TGF-.beta.1)
IT **Stem cell factor**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(growth stimulation of colorectal **carcinoma cells** via the **c-kit** receptor inhibition by TGF-.beta.1)
IT Transforming growth factor .beta. type I receptors
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(growth stimulation of colorectal **carcinoma** cells via the **c-kit** receptor inhibition by TGF-.beta.1)
IT Transforming growth factor .beta. type II receptors
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(growth stimulation of colorectal **carcinoma** cells via the **c-kit** receptor inhibition by TGF-.beta.1)
IT **c-kit** gene (animal)
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(growth stimulation of colorectal **carcinoma** cells via the **c-kit** receptor inhibition by TGF-.beta.1)
IT **c-Kit** (protein)
RL: BPR (Biological process); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)

(growth stimulation of colorectal **carcinoma** cells via the **c-kit** receptor inhibition by TGF-.beta.1)
IT Transforming growth factor .beta. receptors
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(type III; growth stimulation of colorectal **carcinoma** cells via the **c-kit** receptor inhibition by TGF-.beta.1)

L67 ANSWER 19 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1997:413373 HCAPLUS
DN 127:79429
TI **Gain**-of-function mutation of **c-kit** gene in human **gastrointestinal stromal** tumors
AU Hirota, Seiichi; Kitamura, Yukihiko
CS Igakubu, Osaka Daigaku, Suita, 565, Japan
SO Mol. Med. (Tokyo) (1997), 34(6), 698-704
CODEN: MOLMEL; ISSN: 0918-6557
PB Nakayama Shoten
DT Journal; General Review
LA Japanese
CC 14-0 (Mammalian Pathological Biochemistry)
AB A review with 27 refs. Constitutive activation of **c-kit** receptor without binding to its ligand, **stem cell factor** (**SCF**), induces carcinogenesis of mast **cells**. The functions of **C-kit** are reported. Interstitial **cells** of Cajal require **SCF-C-kit** system for differentiation and proliferation, and **c-kit** receptor expression is detected on the **cells**. Function-gaining type mutations occurs in **c-kit** in **gastrointestinal stromal** tumors (GIST).
ST review **ckit** receptor mutation **gastrointestinal** neoplasm
IT **Digestive** system tumors
Mutation
(**c-kit** gene gain-of-function mutation in human **gastrointestinal stromal** tumors)
IT **Stem cell factor**
RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**c-kit** gene gain-of-function mutation in human **gastrointestinal stromal** tumors)
IT **c-kit** gene (animal)
RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence); BPR (Biological process); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**c-kit** gene gain-of-function mutation in human **gastrointestinal stromal** tumors)
IT **c-Kit** (protein)
RL: BOC (Biological occurrence); BPR (Biological process); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**c-kit** gene gain-of-function mutation in human **gastrointestinal stromal** tumors)

L67 ANSWER 20 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1996:587928 HCAPLUS
DN 125:237980
TI Effects of **cyclosporin** A and **FK-506** on **stem cell factor**-induced **histamine** secretion and growth of human mast **cells**
AU Sperr, Wolfgang R.; Agis, Hermine; Czerwenka, Klaus; Virgolini, Irene; Bankl, Hans C.; Muller, Michael R.; Zsebo, Krisztina; Lechner, Klaus; Valent, Peter
CS Department Internal I, University Vienna, Vienna, A-1090, Austria
SO J. Allergy Clin. Immunol. (1996), 98(2), 389-399
CODEN: JACIBY; ISSN: 0091-6749

DT Journal
LA English
CC **1-7** (Pharmacology)
AB **Stem cell factor** (**SCF**) is a key regulator of human mast **cells** (MCs) and a potential mediator of **allergy**. In this study the effects of **cyclosporin** A (CSA) and **FK-506**, two potent immunosuppressive drugs, on **SCF**-dependent **histamine** release and growth of human MCs were analyzed. Preincubation of tissue MCs with CSA (3 .mu.g/mL) resulted in inhibition of **histamine** release provoked by either recombinant human (rh) **SCF** (70.3% .+-. 20.6% inhibition) or anti-IgE (76.7% .+-. 21.9%) or by rhSCF+ anti-IgE (77.4% .+-. 13.9%). Almost the same inhibition was produced by **FK-506** (rhSCF: 82.0% .+-. 18.9% inhibition,; anti-IgE: 71.5% .+-. 16.7%,; rhSCF+ anti-IgE: 70.0% .+-. 7.3%). The effects of CSA and **FK-506** on **SCF**-dependent release of **histamine** were dose-dependent (IC50: CSA, 1 to 10 ng/mL; **FK-506**, 0.3 to 3 ng/mL). IC50 values about three to 10 times higher were found for MCs preincubated with rhSCF before anti-IgE activation, compared with anti-IgE or **SCF** alone. **SCF**-dependent differentiation of human MCs was analyzed in a long-term suspension culture system. Unexpectedly, CSA and **FK-506** were unable to suppress, but even enhanced **SCF**-dependent growth of MCs and formation of MC tryptase in long-term culture. Together, CSA and **FK-506** inhibit **SCF**-dependent release of **histamine** from human MCs and even augment **SCF**-dependent growth of human MCs in long-term culture.
ST **cyclosporin** A FK506 **stem cell factor** ; mast **cell cyclosporin** A FK506
IT Immunosuppressants
Mast **cell**
(effects of **cyclosporin** A and **FK-506** on **stem cell factor**-induced **histamine** secretion and growth of human mast **cells**)
IT Hemopoietins
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(hematopoietic **cell** growth **factors** KL, effects of **cyclosporin** A and **FK-506** on **stem cell factor**-induced **histamine** secretion and growth of human mast **cells**)
IT 59865-13-3, **Cyclosporin** A 104987-11-3, **FK-506**
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(effects of **cyclosporin** A and **FK-506** on **stem cell factor**-induced **histamine** secretion and growth of human mast **cells**)
IT 51-45-6, **Histamine**, biological studies
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(effects of **cyclosporin** A and **FK-506** on **stem cell factor**-induced **histamine** secretion and growth of human mast **cells**)

L67 ANSWER 21 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1996:390429 HCAPLUS
DN 125:77323
TI Interaction of **stem cell factor** and its receptor **c-kit** mediates lodgment and acute expansion of hematopoietic **cells** in the murine spleen
AU Broudy, Virginia C.; Lin, Nancy L.; Priestley, Gregory V.; Nocka, Karl; Wolf, Norman S.
CS Division of Hematology, University of Washington, Seattle, WA, 98195, USA
SO Blood (1996), 88(1), 75-81
CODEN: BLOOAW; ISSN: 0006-4971
DT Journal

LA English
CC 2-10 (Mammalian Hormones)
AB The phenotypes of mice that harbor a defect in the genes encoding either **stem cell factor** (**SCF**) or its receptor, **c-kit**, indicate that this ligand/receptor pair is necessary for maintenance of normal hematopoiesis in the adult. The objective was to det. whether **SCF**, like erythropoietin, is necessary for acute erythroid expansion during recovery from hemolytic anemia. Monoclonal antibody **ACK2**, which recognizes the murine **c-kit** receptor, was used to selectively block the hematopoietic growth-promoting effects of **SCF**. Mice were treated with phenylhydrazine on day 0 and day 1 to induce hemolytic anemia and also received no antibody, control IgG, or **ACK2** on day 0. The mice were killed on day 3 and the hematocrit (Hct), reticulocyte count, and nos. of erythroid and myeloid hematopoietic progenitor **cells** (colony-forming unit-erythroid [CFU-E], burst-forming unit [BFU]-E, and CFU-granulocyte-macrophage [GM]) were quantitated in the femoral marrow and spleen using hematopoietic colony-forming assays. Induction of hemolytic anemia with phenylhydrazine resulted in a drop in the Hct from approx. 50-30%, and an approx. 8-10-fold increase in the reticulocyte count. The nos. of CFU-E increased modestly in the femur, and approx. 25-50-fold in the spleen, in comparison with normal mice. BFU-E and CFU-GM values did not increase in the femur but expanded 6-10-fold in the spleen, in comparison with normal mice. This confirms that much of the erythroid expansion in response to hemolytic anemia occurs in the murine spleen. Neutralizing quantities of the **ACK2** antibody reduced femoral CFU-E, BFU-E, and CFU-GM content to less than half that found in phenylhydrazine-treated control mice and nearly totally ablated splenic hematopoiesis. These results suggest that **c-kit** receptor function may be required for optimal response to acute erythropoietic demand and that erythropoiesis in the splenic microenvironment is more dependent on **SCF/c-kit** receptor interaction than is erythropoiesis in the marrow microenvironment. Because expansion of late erythropoiesis in the spleen was preferentially blocked, the authors tested the hypothesis that homing of more primitive hematopoietic **cells** to the spleen was dependent on **c-kit** receptor function. Lethally irradiated mice were injected with marrow **cells** obtained from mice that had received phenylhydrazine plus control IgG or with marrow **cells** obtained from mice that had received phenylhydrazine plus **ACK2**. In parallel expts., normal murine marrow **cells** were treated in vitro with control IgG or with **ACK2** and were treated in vitro with control IgG or with **ACK2** and were injected into lethally irradiated mice. The fraction of BFU-E and CFU-GM retrieved from the marrow and spleen of the recipient mice 4 h later was reduced by .apprx.75% when progenitor **cells** had been exposed to **ACK2**, in comparison with control IgG. Apparently, interaction of **SCF** with the **c-kit** receptor affects the homing behavior of hematopoietic progenitor **cells** in the adult animal.
ST **stem cell factor** hematopoiesis spleen; **ckit** receptor hematopoiesis spleen
IT Erythropoiesis
Hematocrit
Hematopoiesis
Reticulocyte
Spleen
(interaction of **stem cell factor** and its receptor **c-kit** mediates lodgment and acute expansion of hematopoietic **cells** in murine spleen)
IT Hematopoietic precursor **cell**
(erythroid burst-forming, interaction of **stem cell factor** and its receptor **c-kit** mediates lodgment and acute expansion of hematopoietic **cells** in murine spleen)
IT Hematopoietic precursor **cell**
(erythroid colony-forming, interaction of **stem cell**

factor and its receptor **c-kit** mediates lodgment and acute expansion of hematopoietic **cells** in murine spleen)

IT Hematopoietic precursor **cell**
(granulocyte-macrophage, interaction of **stem cell factor** and its receptor **c-kit** mediates lodgment and acute expansion of hematopoietic **cells** in murine spleen)

IT Hemopoietin receptors
Receptors
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(hematopoietic **cell** growth **factor** KL, interaction of **stem cell factor** and its receptor **c-kit** mediates lodgment and acute expansion of hematopoietic **cells** in murine spleen)

IT Hemopoietins
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(hematopoietic **cell** growth **factors** KL, interaction of **stem cell factor** and its receptor **c-kit** mediates lodgment and acute expansion of hematopoietic **cells** in murine spleen)

L67 ANSWER 22 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1996:372170 HCAPLUS
DN 125:55286
TI **Recombinant** human **stem cell factor** (kit ligand) promotes human **mast cell** and **melanocyte** hyperplasia and functional activation in vivo
AU Costa, John J.; Demetri, George D.; Harrist, Terence J.; Dvorak, Ann M.; Hayes, Daniel F.; Merica, Elizabeth A.; Menchaca, Dora M.; Gringeri, Anthony J.; Schwartz, Lawrence B.; Galli, Stephen J.
CS Dep. Pathology and Med., Beth Israel Hosp. and Harvard Med. Sch., Boston, MA, 02215, USA
SO J. Exp. Med. (1996), 183(6), 2681-2686
CODEN: JEMEAV; ISSN: 0022-1007
DT Journal
LA English
CC 14-9 (Mammalian Pathological Biochemistry)
AB **Stem cell factor** (**SCF**), also known as **mast cell** growth **factor**, kit ligand, and Steel **factor**, is the ligand for the **tyrosine kinase** receptor (SCFR) that is encoded by the c-kit proto-oncogene. We analyzed the effects of recombinant human **SCF** (r-hSCF, 5-50 .mu.g/kg/day, injected s.c.) on **mast cells** and **melanocytes** in a phase I study of 10 patients with advanced breast carcinoma. A wheal and flare reaction developed at each r-hSCF injection site; by electron microscopy, most dermal **mast cells** at these sites exhibited extensive, **anaphylactic** -type degranulation. A 14-d course of r-hSCF significantly increased dermal **mast cell** d. at sites distant to those injected with the cytokine and also increased both urinary levels of the major histamine metabolite, methyl-histamine, and serum levels of **mast cell** .alpha.-tryptase. Five subjects developed areas of persistent **hyperpigmentation** at r-hSCF injection sites; by light microscopy, these sites exhibited markedly increased epidermal **melanization** and increased nos. of **melanocytes**. The demonstration that r-hSCF can promote both the hyperplasia and the functional activation of human **mast cells** and **melanocytes** in vivo has implications for our understanding of the role of endogenous **SCF** in health and disease. These findings also indicate that the interaction between **SCF** and its receptor represents a potential therapeutic target for regulating the nos. and functional activity of both **mast cells** and cutaneous **melanocytes**.
ST **stem cell factor mast cell**

hyperplasia; **melanocyte** hyperplasia **stem cell factor**

IT **Melanocyte**
(disease, hyperplasia; recombinant human **stem cell factor** promotes human **mast cell** and **melanocyte** hyperplasia and functional activation)

IT **Mast cell**
(disease, hyperplasia, recombinant human **stem cell factor** promotes human **mast cell** and **melanocyte** hyperplasia and functional activation)

IT Hemopoietins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(hematopoietic **cell** growth **factors** KL, recombinant human **stem cell factor** promotes human **mast cell** and **melanocyte** hyperplasia and functional activation)

IT **Skin, disease**
(**pigmentation**, recombinant human **stem cell factor** promotes human **mast cell** and **melanocyte** hyperplasia and functional activation)

IT 501-75-7
RL: MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(recombinant human **stem cell factor** promotes human **mast cell** and **melanocyte** hyperplasia and functional activation)

IT 97501-93-4, Tryptase
RL: MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(.alpha.-; recombinant human **stem cell factor** promotes human **mast cell** and **melanocyte** hyperplasia and functional activation)

L67 ANSWER 23 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1996:334134 HCAPLUS
DN 125:31083
TI Expression of **stem-cell factor** and its receptor **c-kit** protein in normal testicular tissue and malignant **germ-cell tumors**
AU Bokemeyer, Carsten; Kuczyk, Markus A.; Dunn, Theresa; Serth, Juergen; Hartmann, Kristin; Jonasson, Jens; Pietsch, Torsten; Jonas, Udo; Schmoll, Hans-Joachim
CS Medical School, Hannover University, Hannover, D-30625, Germany
SO J. Cancer Res. Clin. Oncol. (1996), 122(5), 301-306
CODEN: JCROD7; ISSN: 0171-5216
DT Journal
LA English
CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 2, 13
AB The proto-oncogene **c-kit** and its ligand **stem -cell factor** (**SCF**) may play an important role in the development of normal and malignant testicular tissue. This study investigates the presence of **SCF** and **c-kit** protein in 32 orchiectomy specimens of patients with testicular **cancer**, in 5 specimens of normal testicular tissue and in three established non-seminomatous **germ-cell cancer cell** lines (H12.1, H32, 577ML) by an immunohistochem. approach. Out of 9 testicular **cancer** specimens classified as pure seminomas, 7 (78%) showed a strong immunohistochem. reaction for both **SCF** and **c-kit** protein on the surface of the **tumor cells**. Fourteen non-seminomatous **germ-cell tumors** composed of embryonal **carcinoma** were completely neg. for both **SCF** and **c-kit** protein and only faint positivity was found in 6 **tumors** (26%). Differentiated teratomatous structures within the specimens of non-seminomatous **tumors** showed a strong

immunohistochem. reaction for **SCF** and **c-kit** protein in 8 of 11 (73%) cases. All three testicular **cancer cell** lines showed only faint staining reactions for **c-kit** protein and none for **SCF**. No secretion of **SCF** by the three lines in vitro was detected. The addn. of high concns. of **SCF** (100 ng/mL) to the testicular **cancer cell** lines in culture conditions without fetal calf serum resulted in a 1.4 to 3-fold growth stimulation compared to **cell** growth in serum-free medium alone. This effect was not detectable when the **cells** were cultured in serum-contg. media. In the normal testicular tissue the **germ-cells** displayed a strong immunohistochem. reaction for **c-kit** protein while **SCF** positivity was found at the tubular membrane and on the surface of Sertoli **cells**. The **SCF/c-kit** system may possess a regulatory function in normal testicular tissue by possibly providing the microenvironment necessary for **spermatogenesis**. With the development of testicular **cancer**, this regulatory system seems to be lost, particularly in non-seminomatous **germ-cell tumors**. A growth-stimulatory effect of high concns. of **SCF** on non-seminomatous testicular **cancer cell** lines can be detected only in culture conditions with serum-free media. The effects achievable by the combination of **SCF** with other growth **factors** need to be further studied, as well as the role of the **c-kit/SCF** regulatory system for normal **spermatogenesis** and its possible implications for the understanding and treatment of male **infertility**.

ST **stem cell factor ckit** testis **cancer**

IT **Spermatogenesis**
Testis
Testis, **neoplasm**
(**stem-cell factor** and receptor **c-kit** protein expression in normal human testicular tissue and malignant **germ-cell tumors**)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**c-kit, stem-cell factor** and receptor **c-kit** protein expression in normal human testicular tissue and malignant **germ-cell tumors**)

IT Testis, **neoplasm**
(**germinoma, stem-cell factor** and receptor **c-kit** protein expression in normal human testicular tissue and malignant **germ-cell tumors**)

IT Hemopoietin receptors
Receptors
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(hematopoietic **cell** growth **factor** KL, **stem-cell factor** and receptor **c-kit** protein expression in normal human testicular tissue and malignant **germ-cell tumors**)

IT Hemopoietins
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(hematopoietic **cell** growth **factors** KL, **stem-cell factor** and receptor **c-kit** protein expression in normal human testicular tissue and malignant **germ-cell tumors**)

IT Testis, **neoplasm**
(**seminoma, stem-cell factor** and receptor **c-kit** protein expression in normal human testicular tissue and malignant **germ-cell tumors**)

IT Testis, **neoplasm**
(teratoma, **stem-cell factor** and receptor **c-kit** protein expression in normal human testicular tissue and malignant **germ-cell tumors**)
IT 138359-29-2
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**stem-cell factor** and receptor **c-kit** protein expression in normal human testicular tissue and malignant **germ-cell tumors**)

L67 ANSWER 24 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1996:284892 HCAPLUS
DN 125:77468
TI Effects of **SCF** removal and **ACK2** addition on the induction of apoptosis in cultured mouse neural crest cells
AU Ito, Masaru; Kawa, Yoko; Baba, Takako; Kubota, Yasuo; Mizoguchi, Masako
CS Dep. Dermatol., St. Marianna Univ. Sch. Med., Kawasaki, 216, Japan
SO Nippon Hifuka Gakkai Zasshi (1996), 106(3), 239-248
CODEN: NHKZAD; ISSN: 0021-499X
DT Journal
LA Japanese
CC 2-10 (Mammalian Hormones)
AB The role of **stem cell factor** (**SCF**) in the **c-Kit** expression and melanogenesis in cultured mouse neural crest **cells** was studied. The **c-KIT**-pos. melanoblasts appeared in the **SCF**-added medium when the neural tubes of 9.5-day-old mice embryos were cultivated for 7 days. Using this culture system, we studied whether **ACK2** (monoclonal anti-**c-KIT** antibody) could induce apoptosis in melanoblasts. Apoptotic **cells** of the cultured neural crest **cells** were detected by using Apop Tag kit, and their no./well was counted. We cultured neural tubes in **SCF**-added medium for 7 days, divided them into 5 groups, and cultivated each of them for another 24 h under 1 of the following conditions: **SCF** group, with **SCF**; **SCF**+/- group, without **SCF**; **ACK2** group, with **SCF** and **ACK2**; **ACK2**' group, without **SCF** and with **ACK2**; IgG group, with **SCF** and IgG. There were significantly larger nos. of apoptotic **cells** in **SCF**+/-, **ACK2** and **ACK2**' groups as compared to the **SCF** and IgG groups. The presence of apoptotic **cells** was also confirmed by electron microscopic study. Our in vitro study shows that **ACK2** causes apoptosis in **c-Kit**-pos. melanoblasts and that **SCF** promotes melanocyte survival and differentiation by suppressing apoptosis.
ST **stem cell factor** melanoblast apoptosis
IT Apoptosis
Cell differentiation
Embryo
Melanoblast
Melanocyte
(melanoblast apoptosis suppression by **stem cell factor** and promotion of melanocyte survival and differentiation)
IT Hemopoietin receptors
Receptors
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(hematopoietic **cell** growth **factor** KL, melanoblast apoptosis suppression by **stem cell factor** and promotion of melanocyte survival and differentiation)
IT Hemopoietins
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(hematopoietic **cell** growth **factors** KL, melanoblast apoptosis suppression by **stem cell factor**

and promotion of melanocyte survival and differentiation)
IT Nervous system
(neural crest, melanoblast apoptosis suppression by **stem cell factor** and promotion of melanocyte survival and differentiation)

L67 ANSWER 25 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1995:641162 HCAPLUS
DN 123:132909
TI The **effects** of **stem cell factor**, the ligand for the c-kit receptor, on mouse and human mast **cell** development, **survival**, and function
AU Galli, Stephen J.; Tsai, Mindy; Wershil, Barry K.; Iemura, Akihiko; Ando, Akikazu; Tam, See-Ying; Costa, John J.
CS Department of Pathology, Beth Israel Hospital, Boston, MA, 02215, USA
SO Biol. Mol. Aspects Mast Cell Basophil Differ. Funct. (1995), 1-11. Editor(s): Kitamura, Yukihiko. Publisher: Raven, New York, N. Y. CODEN: 61OBAK
DT Conference; General Review
LA English
CC 2-0 (Mammalian Hormones)
AB A review, with 42 refs., on some of the important effects of **stem cell factor** (**SCF**) in mast **cell** biol., focusing primarily on the results of in vivo studies and on those issues which currently appear to be of clin. relevance in humans. Included were discussions on the identification and characterization of **SCF**; effects of **SCF** in mast **cell** development and survival; promotion of mast **cell** survival by **SCF** by suppressing apoptosis; **SCF** regulation of mast **cell** secretory function and mediatory release; **SCF** effects on IGE-dependent passive **anaphylaxis** in mice; and effects of **SCF** in primates and humans in vivo.
ST review **stem cell factor** mast **cell**
IT Mast **cell**
(**stem cell factor**, effects on mouse and human mast **cell** development and survival and function)
IT Hemopoietins
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(hematopoietic **cell** growth **factors** KL, **stem cell factor**, effects on mouse and human mast **cell** development and survival and function)

L67 ANSWER 26 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1995:245385 HCAPLUS
DN 122:7833
TI **Recombinant stem cell factor** -induced mast **cell** activation and smooth muscle contraction in human **bronchi**
AU Undem, Bradley J.; Lichtenstein, Lawrence M.; Hubbard, Walter C.; Meeker, Sonya; Ellis, James L.
CS Dep. Med., Johns Hopkins Univ., Baltimore, MD, USA
SO Am. J. Respir. Cell Mol. Biol. (1994), 11(6), 646-50 CODEN: AJRBEL; ISSN: 1044-1549
DT Journal
LA English
CC 15-9 (Immunochemistry)
AB The effect of human recombinant **stem cell factor** (**SCF**) on inflammatory mediator release and smooth muscle contraction was evaluated in human isolated intralobar **bronchi**. **Bronchi** from 21 of 26 donors contracted in response to **SCF**. The threshold concn. was approx. 0.01 .mu.g/mL. At 1 .mu.g/mL, the tissues contracted to about 60% of the carbamylcholine-induced max. contraction. The responses to **SCF** mimicked those obtained with anti-IgE. Thus, the contractions to **SCF** and anti-IgE were inhibited to a similar extent by a

combination of a cysteinyl-leukotriene receptor antagonist and a histamine H1 receptor antagonist. **SCF** also mimicked the effect of anti-IgE in releasing histamine, i-LTD4, and PGD2 from the **bronchi**. At a threshold concn. for contraction (0.01 .mu.g/mL), **SCF** had no effect on subsequent responses to anti-IgE in the **bronchi**. Apparently, human recombinant **SCF** contracts airway smooth muscle by stimulating the release of contractile mediators from **bronchial** mast **cells**. The data fail to support the hypothesis that **SCF** primes **bronchial** mast **cells** to subsequent immunol. stimuli.

ST **stem cell factor** mast **cell** **bronchi**

IT **Bronchi**
Mast **cell**
(**stem cell factor**-induced mast **cell** activation and smooth muscle contraction in human **bronchi**)

IT Hemopoietins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(hematopoietic **cell** growth **factors** KL, **stem cell factor**-induced mast **cell** activation and smooth muscle contraction in human **bronchi**)

IT 51-45-6, Histamine, biological studies 41598-07-6, PGD2 73836-78-9, LTD4
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(contractile mediators release from **bronchial** mast **cells** induced by **stem cell factor**)

L67 ANSWER 27 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1994:406366 HCAPLUS
DN 121:6366
TI Coexpression of the **c-kit** receptor and the **stem cell factor** in **gynecological tumors**
AU Inoue, Masaki; Kyo, Satoru; Fujita, Masami; Enomoto, Takayuki; Kondoh, Gen
CS Sch. Med., Osaka Univ., Suita, 565, Japan
SO Cancer Res. (1994), 54(11), 3049-53
CODEN: CNREA8; ISSN: 0008-5472
DT Journal
LA English
CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 13, 15

AB The protooncogene **c-kit** encodes a transmembrane receptor-type tyrosine kinase which belongs to the .beta.-PDGER/CSF-1 receptor tyrosine kinase family. The interaction between **c-kit** receptor and its corresponding ligand, **stem cell factor** (**SCF**), has been suggested to be involved in embryogenesis as well as **carcinogenesis** via the autocrine/paracrine system. In the present study, **cancer cell** lines and normal/benign/malignant tissues of the human female genital tract were examd. for the expression of both **c-kit** and **SCF** by Northern blot and immunohistochem. analyses. Two of 16 **cell** lines showed mRNA expression of both **c-kit** and **SCF**, while 2 and 12 **cell** lines expressed **c-kit** and **SCF**, resp. In tissues, several cases of malignant **tumors**, including three cervical **cancers**, one ovarian **cancer**, and one ovarian immature teratoma, expressed mRNA of both **c-kit** and **SCF**. In normal tissues, squamous epithelium expressed **SCF** immunohistochem., while **c-kit** protein was detected only in melanocytes. Some tissues of malignant **tumors**, one squamous **cell carcinoma** of the cervix, two small **cell carcinomas** of the cervix, two serous **adenocarcinomas** of the ovary, and two immature teratomas of the ovary, expressed both **c-kit** and **SCF** proteins

immunohistochem. It is also notable that **c-kit** protein was expressed only in malignant **germ cells** of dysgerminous, while **SCF** was expressed in the connective tissues surrounding **germ cells**. The present study suggests that the **c-kit/SCF** system may play an important role in the **carcinogenesis** of the female genital tract.

ST gene **ckit** receptor **neoplasm** ovary cervix

IT Melanocyte
(**c-kit** receptor and **stem cell factor** expression in, in human)

IT Ribonucleic acids, messenger
RL: BIOL (Biological study)
(for **c-kit** receptor and **stem cell factor**, in human **gynecol. tumors**)

IT Ovary, **neoplasm**
(**adenocarcinoma**, **c-kit** receptor and **stem cell factor** expression in, in human)

IT **Uterus**
(cervix, epithelium, **c-kit** receptor and **stem cell factor** expression in, in human)

IT **Uterus, neoplasm**
(cervix, small-**cell carcinoma**, **c-kit** receptor and **stem cell factor** expression in, in human)

IT **Uterus, neoplasm**
(cervix, squamous **cell carcinoma**, **c-kit** receptor and **stem cell factor** expression in, in human)

IT Ovary, **neoplasm**
(**dysgerminoma**, **c-kit** receptor and **stem cell factor** expression in, in human)

IT Hemopoietin receptors
Receptors
RL: PROC (Process)
(hematopoietic **cell** growth **factor** KL, expression of, in human **gynecol. tumors**, **stem cell factor** in relation to)

IT Hemopoietins
RL: BIOL (Biological study)
(hematopoietic cell growth factors KL, expression of, in human **gynecol. tumors**, **c-kit** receptor in relation to)

IT Ovary, **neoplasm**
(teratoma, **c-kit** receptor and **stem cell factor** expression in, in human)

IT Reproductive tract
(vulva, epithelium of, **c-kit** receptor and **stem cell factor** expression in, in human)

L67 ANSWER 28 OF 30 HCAPLUS COPYRIGHT 2000 ACS

AN 1994:183929 HCAPLUS

DN 120:183929

TI **Stem cell factor** induces outgrowth of **c-kit**-positive neurites and supports the survival of **c-kit**-positive neurons in dorsal root ganglia of mouse embryos

AU Hirata, Tatsumi; Morii, Eiichi; Morimoto, Masahiro; Kasugai, Tsutomu; Tsujimura, Tohru; Hirota, Seiichi; Kanakura, Yuzuru; Nomura, Shintaro; Kitamura, Yukihiko

CS Med. Sch., Osaka Univ., Suita, 565, Japan

SO Development (Cambridge, UK) (1993), 119(1), 49-56
CODEN: DEVPED; ISSN: 0950-1991

DT Journal

LA English

CC 2-10 (Mammalian Hormones)

AB The **c-kit** receptor tyrosine kinase is highly expressed by about 10% of the neurons in the dorsal root ganglia (DRGs) of mouse embryos. The authors investigated the in vitro effect of **stem cell factor** (**SCF**), the ligand for **c-kit** receptor, on DRGs. Recombinant murine **SCF** (rmSCF) induced the outgrowth of **c-kit**-pos. neurites from DRGs of normal (+/+) embryos. The effect of **SCF** was dose dependent and completely abolished by anti-**c-kit ACK2** monoclonal antibody (mAb). Some neurites whose outgrowth was induced by NGF were **c-kit**-pos., but anti-NGF mAb did not inhibit the rmSCF-induced neurite outgrowth. The rmSCF did not induce neurite outgrowth from DRGs of W/W embryos that did not express **c-kit** receptors on the **cell** surface and of W42/W42 mutant embryos that expressed **c-kit** receptors without tyrosine kinase activity. The rmSCF also had a trophic effect on **c-kit**-pos. neurons in the culture of dissocd. DRG **cells**. Most **c-kit**-pos. neurons appeared to respond to NGF as well, and the **SCF**-responsive subpopulation represented about 10% of NGF-responsive neurons. The rmSCF did not support the survival of DRG neurons from embryos of W/W and W42/W42 genotypes. These results suggest that the stimulus through the **c-kit** receptor tyrosine kinase has an important role in development of the peripheral nervous system.

ST **stem cell factor** nerve **c-kit**; embryo nerve **stem cell factor**

IT Embryo
(nerve response to **stem cell factor** in dorsal root ganglia of, gene **c-kit** receptor tyrosine kinase in relation to)

IT Nerve
(**stem cell factor** neurotrophic action on, in dorsal root ganglia of embryo, gene **c-kit** receptor tyrosine kinase in relation to)

IT Nerve
(axon, outgrowth of, **stem cell factor** induction of, in dorsal root ganglia of embryo, gene **c-kit** receptor tyrosine kinase in relation to)

IT Receptors
RL: BIOL (Biological study)
(hematopoietic **cell** growth **factor** KL, nerve response to **stem cell factor** in dorsal root ganglia of embryo in relation to)

IT Hemopoietins
RL: BIOL (Biological study)
(hematopoietic cell growth factors KL, nerve response to, in dorsal root ganglia of embryo, gene **c-kit** receptor tyrosine kinase in relation to)

IT Hemopoietins
RL: BIOL (Biological study)
(hematopoietic **cell** growth **factors** KL, receptors, nerve response to **stem cell factor** in dorsal root ganglia of embryo in relation to)

IT Nerve center and Ganglion
(spinal, nerve response to **stem cell factor** in, of embryo, gene **c-kit** receptor tyrosine kinase in relation to)

IT 138359-29-2, **c-Kit** receptor tyrosine kinase
RL: BIOL (Biological study)
(nerve response to **stem cell factor** in dorsal root ganglia of embryo in relation to)

IT 9061-61-4, Nerve growth **factor**
RL: BIOL (Biological study)
(nerve response to, in dorsal root ganglia of embryo, **stem cell factor** in relation to)

L67 ANSWER 29 OF 30 HCAPLUS COPYRIGHT 2000 ACS

AN 1994:161208 HCAPLUS
DN 120:161208
TI Possible role of **stem cell factor** as a serum **factor**: monoclonal anti-**c-kit** antibody abrogates interleukin-6-dependent colony growth in serum-containing culture
AU Shiohara, Masaaki; Koike, Kenichi; Kubo, Tetsuo; Amano, Yoshiro; Takagi, Mineo; Muraoka, Kenji; Nakao, Junji; Nakahata, Tatsutoshi; Komiyama, Atsushi
CS Sch. Med., Shinshu Univ., Matsumoto, 390, Japan
SO Exp. Hematol. (Charlottesville, Va.) (1993), 21(7), 907-12
CODEN: EXHMA6; ISSN: 0301-472X
DT Journal
LA English
CC 15-5 (Immunochemistry)
Section cross-reference(s): 2
AB The monoclonal rat anti-**c-kit** antibody (**ACK2**), which abrogates colony growth supported by **stem cell factor** (**SCF**), significantly inhibited the interleukin-6 (IL-6)-dependent growth of hematopoietic progenitors derived from spleen **cells** of normal and 5-fluorouracil (5-FU)-treated mice and from bone marrow **cells** of normal mice in serum-contg. culture. The nos. and types of colonies supported by IL-3, granulocyte-macrophage colony-stimulating **factor** (GM-CSF) and granulocyte colony-stimulating **factor** (G-CSF), however, were not influenced by the addn. of **ACK2** to the cultures of the bone marrow **cells** from normal mice. In replating expts. with pooled blast **cells**, **ACK2** caused a partial, but significant, inhibition of GM colony growth supported by a combination of IL-6 and fetal bovine serum (FBS), which suggests that FBS is one source of the **SCF** activity. Conversely, the addn. of **SCF** or FBS with IL-6 to a serum-free culture had significant synergistic effects on the total no. of colonies derived from post-5-FU spleen **cells** and from pooled blast **cells**. The dose response study showed that the ability of 30% FBS to interact with IL-6 on the colony growth by post-5-FU spleen **cells** was equiv. to that of approx. 5 ng/mL **SCF**. These findings suggest that **c-kit** plays an important role in the growth of hematopoietic progenitors responding to IL-6, and that **SCF** in the serum affects the development of hematopoietic progenitors in serum-contg. cultures.
ST serum **stem cell factor** colony growth; interleukin 6 colony growth serum **factor**
IT Hematopoietic precursor **cell**
(**stem cell factor** in blood serum and interleukin 6 effect on colony growth by)
IT Animal tissue culture
(**stem cell factor** in blood serum and interleukin 6 effects on colony growth by hematopoietic precursor **cells** in)
IT Blood serum
(**stem cell factor** in, hematopoietic progenitor **cells** response to interleukin 6 and)
IT Hemopoietins
RL: BIOL (Biological study)
(hematopoietic cell growth factors KL, of blood serum, hematopoietic progenitor cells response to interleukin 6 and)
IT Lymphokines and Cytokines
RL: BIOL (Biological study)
(interleukin 6, **stem cell factor** in blood serum and, colony growth by hematopoietic precursor **cells** response to)

L67 ANSWER 30 OF 30 HCAPLUS COPYRIGHT 2000 ACS
AN 1994:6791 HCAPLUS
DN 120:6791
TI Production, purification and uses of **stem cell**

proliferation **factor** (SCPF)
IN Lawman, Michael J. P.; Bagwell, Charles E.; Lawman, Patricia D.
PA University of Florida Research Foundation Inc., USA
SO PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C12N015-00
ICS C12N015-02; C12N005-22; C12N001-00; C07K015-28; A61K039-395; C07H015-00; G01N033-543
CC 15-5 (Immunochemistry)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9320197	A1	19931014	WO 1993-US3197	19930406
	W: AT, AU, BB, BG, BR, CA, CH, CZ, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SK, UA, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9340467	A1	19931108	AU 1993-40467	19930406
	CN 1081715	A	19940209	CN 1993-105217	19930406
	EP 672128	A1	19950920	EP 1993-911591	19930406
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	JP 07508640	T2	19950928	JP 1993-517758	19930406
	ZA 9302489	A	19940217	ZA 1993-2489	19930407
	AU 9745294	A1	19980212	AU 1997-45294	19971120
	AU 714492	B2	20000106		
PRAI	US 1992-863889		19920406		
	WO 1993-US3197		19930406		

AB The autocrine growth **factor** is isolated from a human **germ cell tumor** line of neuroectodermal origin. The protein exists in two forms, i.e. a sol. form of 32 kDa polypeptide and a membrane bound form of 37 kDa polypeptide. The **factor** stimulates proliferation of human bone marrow **stem cells** and has synergistic effect with other growth **factors**, e.g. interleukin 3, interleukin 6, and **SCF** (**c-kit** oncogene product). Prodn. of SCPF by genetic engineering technique is also claimed. Polyclonal and monoclonal antibody are prepd. for detecting and treating SCPF-assocd. disorders, e.g. leukemia, aplastic anemia, neuronal disorder, severe combined immunodeficiency, and hypersplenism.

ST **stem cell** proliferation **factor**; polyclonal monoclonal antibody leukemia; aplastic anemia immunodeficiency hypersplenism treatment antibody; bone marrow **stem cell** stimulation

IT Animal **cell** line
(of **germ cell tumor**, **stem cell** proliferation **factor** purifn. from)

IT Genetic engineering
(prodn. of **stem cell** proliferation **factor** by)

IT Lymphokines and Cytokines
RL: BIOL (Biological study)
(**stem cell** proliferation **factor**, isolation and prodn. and uses of)

IT Leukemia
Nerve, disease
(**stem cell** proliferation **factor**-assocd., detection and treatment of, antibody to **stem cell** proliferation **factor** for)

IT Antibodies
RL: PREP (Preparation)
(to **stem cell** proliferation **factor**, prepn. of, for detecting and treating leukemia, etc.)

IT Anemia (disease)

(aplastic, **stem cell** proliferation **factor**
-assocd., detection and treatment of, antibody to **stem cell** proliferation **factor** for)
IT Gene, animal
RL: BIOL (Biological study)
(**c-kit**, protein product of, synergism of **stem cell** proliferation **factor** and, for stimulating bone marrow **stem cells** proliferation)
IT Spleen, disease
(hypersplenism, **stem cell** proliferation **factor**-assocd., detection and treatment of, antibody to **stem cell** proliferation **factor** for)
IT Lymphokines and Cytokines
RL: BIOL (Biological study)
(interleukin 3, synergism of **stem cell** proliferation **factor** and, for stimulating bone marrow **stem cells** proliferation)
IT Lymphokines and Cytokines
RL: BIOL (Biological study)
(interleukin 6, synergism of **stem cell** proliferation **factor** and, for stimulating bone marrow **stem cells** proliferation)
IT Antibodies
RL: PREP (Preparation)
(monoclonal, to **stem cell** proliferation **factor**, prepn. of, for detecting and treating leukemia, etc.)
IT Gamete and **Germ cell**
(**neoplasm**, **stem cell** proliferation **factor** purifn. from **cell** line of)
IT Nucleotides, polymers
RL: BIOL (Biological study)
(poly-, encoding gene sequence of **stem cell** proliferation **factor**, for prodn.)
IT Immunodeficiency
(severe combined, **stem cell** proliferation **factor**-assocd., detection and treatment of, antibody to **stem cell** proliferation **factor** for)

=> fil medline

FILE 'MEDLINE' ENTERED AT 10:21:40 ON 28 JUN 2000

FILE LAST UPDATED: 22 JUN 2000 (20000622/UP). FILE COVERS 1960 TO DATE.

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OLDMEDLINE, data from 1960 through 1965 from the Cumulated Index Medicus (CIM), has been added to MEDLINE. See HELP CONTENT for details.

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THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

=> d his l68-

(FILE 'HCAPLUS' ENTERED AT 09:49:49 ON 28 JUN 2000)

FILE 'MEDLINE' ENTERED AT 09:50:27 ON 28 JUN 2000
L68 2619 S STEM CELL FACTOR
L69 56 S L68 AND SKIN+NT/CT
L70 69 S L68 AND SKIN DISEASES+NT/CT

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L71           4 S L68 AND SKIN PIGMENTATION+NT/CT
L72          12 S L68 AND PIGMENTATION+NT/CT
L73           8 S L68 AND SKIN PHYSIOLOGY+NT/CT
L74           9 S L68 AND ASTHMA+NT/CT
L75           4 S L68 AND ANAPHYLAXIS+NT/CT
L76           0 S L68 AND BRONCHIAL SPASM+NT/CT
L77          29 S L68 AND MASTOCYTOSIS+NT/CT
L78           0 S L68 AND URTICARIA+NT/CT
L79          32 S L68 AND HYPERSENSITIVITY+NT/CT
L80          20 S L68 AND (A3. OR C6.)/CT AND STROM?
L81           5 S L68 AND (A3. OR C6.)/CT AND STROMAL CELLS+NT/CT
L82           3 S L80,L81 AND C4./CT
L83          57 S L68 AND GERM CELLS+NT/CT
L84           1 S L83 AND C4./CT
L85          54 S L68 AND "NEOPLASMS, GERM CELL AND EMBRYONAL"+NT/CT
L86          13 S L68 AND ACK2
L87          60 S L68 AND ?DIMER?
L88          39 S L87 AND KIT
L89          36 S L87 AND PROTO ONCOGENE PROTEIN C KIT
L90         108 S L68 AND LIGAND (L) BIND?
L91         111 S L68 AND (PREGNANCY+NT OR FERTILITY+NT OR INFERTILITY+NT OR CO
L92         125 S L68 AND A5./CT
L93         158 S L68 AND EPITHELIAL CELLS+NT/CT
L94          18 S L68 AND (MELANINS+NT OR KERATIN+NT OR KERATINOCYTES+NT)/CT
               E PROTEIN-TYROSINE KINASE/CT
               E E3+ALL/CT
L95         915 S D8./CT AND L68
L96         296 S L95 AND L69-L94
L97         289 S L69-L94,L96 AND STEM CELL FACTOR/CT
L98         448 S L69-L94,L96 AND STEM CELL FACTOR/CN
L99         448 S L97,L98
L100         37 S L99 AND SIGNAL TRANSDUCTION+NT/CT
L101        172 S L69-L94,L96 NOT L99
L102          6 S L101 AND SIGNAL TRANSDUCTION+NT/CT
L103         43 S L100,L102
L104        112 S L99,L101 AND SIGNAL?
L105         69 S L104 NOT L103
L106         17 S L105 AND PATHWAY
L107          3 S L105 AND  (DOWNSTREAM? OR UPSTREAM?)
L108          1 S L106 AND LUNG NEOPLASMS/CT
L109         44 S L103,L108
L110        687 S L68 AND (AI/CT OR INHIBIT? OR BLOCK? OR ANTAGON?)
L111        166 S L110 AND L99,L101
L112          5 S L111 AND ((STEM CELL FACTOR)(L)AI)/CT
L113         47 S L109,L112

     FILE 'MEDLINE' ENTERED AT 10:21:40 ON 28 JUN 2000

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L113 ANSWER 1 OF 47  MEDLINE
AN   2000134033     MEDLINE
DN   20134033
TI   c-Kit and c-kit mutations in mastocytosis and other
     hematological diseases.
AU   Boissan M; Feger F; Guillosson J J; Arock M
CS   Cellular and Molecular Hematology Unit, Faculty of Pharmacy, Paris,
     France.
SO   JOURNAL OF LEUKOCYTE BIOLOGY, (2000 Feb) 67 (2) 135-48.  Ref: 129
     Journal code: IWY. ISSN: 0741-5400.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
     General Review; (REVIEW)
     (REVIEW, ACADEMIC)
LA   English
```

FS Priority Journals; Cancer Journals
EM 200004
EW 20000404
AB Mast cells (MC) are tissue elements derived from hematopoietic stem cells. Their differentiation and proliferation processes are under the influence of cytokines, including one of utmost importance known as **stem cell factor** (SCF). SCF receptor is encoded by the protooncogene c-**kit**, belongs to the type III receptor tyrosine kinase subfamily, and is also expressed on other hematopoietic or non-hematopoietic cells. Ligation of c-**kit** receptor by SCF induces its **dimerization**, followed by induction of multiple intracellular signaling pathways leading to cell proliferation and activation. Mastocytosis, a relatively rare group of diseases characterized by accumulation of MC in various tissues, are found isolated or sometimes associated with other hematological malignancies in humans. Although the initial events leading to mastocytosis are not yet unraveled, alterations of the c-**kit** gene have been described. Particularly interesting are acquired mutations resulting in a constitutively activated receptor, possibly involved in the increased numbers of MC in tissues. For this reason, future strategies might be envisaged to target specifically the mutated c-**kit** and/or its intracellular signaling.
CT Check Tags: Animal; Human
Amino Acid Substitution
Cell Differentiation
Cell Division
Cell Transformation, Neoplastic
Dimerization
Hematologic Diseases: GE, genetics
*Hematologic Diseases: ME, metabolism
Hematologic Neoplasms: GE, genetics
Hematologic Neoplasms: ME, metabolism
Leukemia: GE, genetics
Leukemia: ME, metabolism
Mastocytosis: GE, genetics
***Mastocytosis: ME, metabolism**
Mice
Neoplasm Proteins: GE, genetics
Neoplasm Proteins: PH, physiology
Phosphorylation
Point Mutation
Protein Processing, Post-Translational
Protein Structure, Tertiary
Proto-Oncogene Protein c-kit: CH, chemistry
Proto-Oncogene Protein c-kit: GE, genetics
***Proto-Oncogene Protein c-kit: PH, physiology**
Proto-Oncogenes
Rats
Sequence Deletion
Signal Transduction
Stem Cell Factor: PH, physiology
Tumor Cells, Cultured
CN EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); 0 (Neoplasm Proteins); **0 (Stem Cell Factor)**

L113 ANSWER 2 OF 47 MEDLINE
AN 2000120715 MEDLINE
DN 20120715
TI Kit/**stem cell factor** receptor-induced activation of phosphatidylinositol 3'-kinase is essential for male fertility.
AU Blume-Jensen P; Jiang G; Hyman R; Lee K F; O'Gorman S; Hunter T
CS Molecular Biology and Virology Laboratory, The Salk Institute, La Jolla, California, USA.. blume@salk.edu
SO NATURE GENETICS, (2000 Feb) 24 (2) 157-62.
Journal code: BRO. ISSN: 1061-4036.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200005
EW 20000501
AB The c-kit-encoded transmembrane tyrosine kinase receptor for **stem cell factor** (Kit/SCF-R) is required for normal haematopoiesis, melanogenesis and gametogenesis. However, the roles of individual Kit/SCF-R-induced signalling pathways in the control of developmental processes in the intact animal are completely unknown. To examine the function of SCF-induced phosphatidylinositol (PI) 3'-kinase activation in vivo, we employed the Cre-loxP system to mutate the codon for Tyr719, the PI 3'-kinase binding site in Kit/SCF-R, to Phe in the genome of mice by homologous recombination. Homozygous (Y719F/Y719F) mutant mice are viable. The mutation completely disrupted PI 3'-kinase binding to Kit/SCF-R and reduced SCF-induced PI 3'-kinase-dependent activation of Akt by 90%. The mutation induced a gender- and tissue-specific defect. Although there are no haematopoietic or pigmentation defects in homozygous mutant mice, males are sterile due to a block in spermatogenesis, with initially decreased proliferation and subsequent extensive apoptosis occurring at the spermatogonial stem-cell level. In contrast, female homozygotes are fully fertile. This is the first report so far demonstrating the role of an individual signalling pathway downstream of Kit/SCF-R in the intact animal. It provides the first in vivo model for male sterility caused by a discrete signalling pathway defect affecting early germ cells.
CT Check Tags: Animal; Female; Male; Support, U.S. Gov't, P.H.S.
Amino Acid Substitution
Apoptosis
Codon
Enzyme Activation
Exons
***Fertility: GE, genetics**
Fetal Development
Genomic Library
Heterozygote
Homozygote
Introns
Mice
Mice, Mutant Strains
Mutagenesis, Site-Directed
Proto-Oncogene Protein c-kit: CH, chemistry
***Proto-Oncogene Protein c-kit: GE, genetics**
***Proto-Oncogene Protein c-kit: ME, metabolism**
Signal Transduction: DE, drug effects
Stem Cell Factor: PD, pharmacology
Stem Cell Factor: PH, physiology
***1-Phosphatidylinositol 3-Kinase: ME, metabolism**
CN EC 2.7.1.137 (1-Phosphatidylinositol 3-Kinase); EC 2.7.11.- (Proto-Oncogene Protein c-kit); 0 (Codon); **0 (Stem Cell Factor)**

L113 ANSWER 3 OF 47 MEDLINE
AN 2000076250 MEDLINE
DN 20076250
TI Distinct signals control the hematopoiesis of lymphoid-related dendritic cells.
AU Galy A; Christopherson I; Ferlazzo G; Liu G; Spits H; Georgopoulos K
CS Barbara Ann Karmanos Cancer Institute, Wayne State University, Detroit, MI 48201, USA.. galya@kci.wayne.edu
SO BLOOD, (2000 Jan 1) 95 (1) 128-37.
Journal code: A8G. ISSN: 0006-4971.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals

EM 200004
EW 20000401
AB The molecular and cellular requirements for the development of different populations of human dendritic cells (DC) were studied. Conditions were defined that support DC production from lymphoid progenitors but that fail to induce DC formation from peripheral monocytes. The production of these lymphoid-related DC was severely blocked when hematopoietic progenitors overexpressed Ik7, a mutant dominant-negative Ikaros protein. In contrast, Ik7 did not block the formation of DC in conditions supporting the development of monocyte-derived DC. Furthermore, Ik7 did not block the formation of monocyte/macrophages and enhanced granulopoiesis. One of the molecular mechanisms mediated by Ik7 appears to be down-regulation of the flt3-receptor mRNA. Thus, distinct signals control the formation of DC demonstrating that some aspects of DC diversity are determined in part by distinct molecular cues at the hematopoietic level. (Blood. 2000;95:128-137)
CT Check Tags: Human; Support, Non-U.S. Gov't
Adult
Antigens, CD: AN, analysis
Antigens, CD34: AN, analysis
Bone Marrow Cells: CY, cytology
Cell Differentiation: DE, drug effects
Cells, Cultured
*Cytokines: PD, pharmacology
Dendritic Cells: CY, cytology
Dendritic Cells: DE, drug effects
***Dendritic Cells: PH, physiology**
Down-Regulation (Physiology)
Flow Cytometry
Granulocyte Macrophage Colony-Stimulating Factors, Recombinant: PD, pharmacology
Hematopoiesis: DE, drug effects
*Hematopoiesis: PH, physiology
Hematopoietic Stem Cells: CY, cytology
Hematopoietic Stem Cells: DE, drug effects
*Hematopoietic Stem Cells: PH, physiology
Interleukins: PD, pharmacology
Lymphocytes: CY, cytology
Lymphocytes: DE, drug effects
*Lymphocytes: PH, physiology
Macrophages: CY, cytology
Monocytes: CY, cytology
Neprilysin: AN, analysis
Proto-Oncogene Proteins: GE, genetics
Receptor Protein-Tyrosine Kinases: GE, genetics
Receptors, Cell Surface: GE, genetics
Recombinant Proteins: ME, metabolism
Recombinant Proteins: PD, pharmacology
Reverse Transcriptase Polymerase Chain Reaction
***Signal Transduction**
Stem Cell Factor: PD, pharmacology
T-Lymphocytes: CY, cytology
T-Lymphocytes: DE, drug effects
*T-Lymphocytes: PH, physiology
Transcription Factors: GE, genetics
Transcription Factors: ME, metabolism
Tumor Necrosis Factor: PD, pharmacology
Zinc Fingers
RN 148971-36-2 (Ikaros protein)
CN EC 2.7.1.- (fetal liver kinase-2); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); EC 3.4.24.11 (Neprilysin); 0 (Antigens, CD); 0 (Antigens, CD34); 0 (Cytokines); 0 (Granulocyte Macrophage Colony-Stimulating Factors, Recombinant); 0 (Interleukins); 0 (Proto-Oncogene Proteins); 0 (Receptors, Cell Surface); 0 (Recombinant Proteins); **0 (Stem Cell Factor)**; 0 (Transcription Factors); 0 (Tumor Necrosis Factor)

L113 ANSWER 4 OF 47 MEDLINE
AN 2000059822 MEDLINE
DN 20059822
TI **Stem cell factor** protects germ cells from apoptosis in vitro.
AU Yan W; Suominen J; Toppari J
CS Departments of Physiology and Pediatrics, University of Turku, Kiinamyllynkatu 10, Turku, Finland.
SO JOURNAL OF CELL SCIENCE, (2000 Jan) 113 (Pt 1) 161-8.
Journal code: HNK. ISSN: 0021-9533.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200005
EW 20000501
AB **Stem cell factor** (SCF) plays an important role in migration, adhesion, proliferation, and survival of primordial germ cells and spermatogonia during testicular development. However, the function of SCF in the adult testis is poorly described. We have previously shown that, in the presence of SCF, there were more type A spermatogonia incorporating thymidine at stage XII of rat seminiferous tubules cultured in vitro than in the absence of SCF, implying that the increased DNA synthesis might result from enhanced survival of spermatogonia. To explore the potential pro-survival function of SCF during spermatogenesis, the seminiferous tubules from stage XII were cultured in the presence or absence of SCF (100 ng/ml) for 8, 24, 48, and 72 hours, respectively, and apoptosis was analyzed by DNA laddering and in situ 3'-end labeling (ISEL) staining. Surprisingly, not only spermatogonia, but also spermatocytes and spermatids, were protected from apoptosis in the presence of SCF. Apoptosis took place much later and was less severe in the SCF-treated tubules than in the controls. Based on previous studies showing that FSH prevents germ cells from undergoing apoptosis in vitro, and that SCF level is increased dramatically in response to FSH stimulation, we also tested if the pro-survival effect of FSH is mediated through SCF by using a function-**blocking** monoclonal antibody, ACK-2, to **block** SCF/c-kit interaction. After 24 hours of **blockade**, the protective effect of FSH was partially abolished, as manifested by DNA laddering and ISEL analyses. The present study demonstrates that SCF acts as an important survival factor for germ cells in the adult rat testis and FSH pro-survival effect on germ cells is mediated partially through the SCF/c-kit pathway.
CT Check Tags: Animal; Male; Support, Non-U.S. Gov't
Antibodies, Monoclonal: PD, pharmacology
*Apoptosis: DE, drug effects
Cell Survival: DE, drug effects
DNA: BI, biosynthesis
DNA: GE, genetics
DNA Fragmentation: DE, drug effects
FSH: PD, pharmacology
In Situ Nick-End Labeling
Protein Binding: DE, drug effects
Proto-Oncogene Protein c-kit: ME, metabolism
Rats
Rats, Sprague-Dawley
Seminiferous Tubules: CY, cytology
Seminiferous Tubules: DE, drug effects
Seminiferous Tubules: GD, growth & development
Seminiferous Tubules: ME, metabolism
Signal Transduction: DE, drug effects
***Spermatozoa: CY, cytology**
***Spermatozoa: DE, drug effects**
Spermatozoa: ME, metabolism
Stem Cell Factor: AI, antagonists & inhibitors
***Stem Cell Factor: PD, pharmacology**
Time Factors

Tissue Culture
RN 9002-68-0 (FSH); 9007-49-2 (DNA)
CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); 0 (Antibodies, Monoclonal); **0 (Stem Cell Factor)**

L113 ANSWER 5 OF 47 MEDLINE
AN 2000049052 MEDLINE
DN 20049052
TI Early signaling pathways activated by c-**Kit** in hematopoietic cells.
AU Linnekin D
CS Basic Research Laboratory, National Cancer Institute-Frederick Cancer Research and Development Center, MD 21702-1201, USA.. dlinnekin@mail.ncifcrf.gov
SO INTERNATIONAL JOURNAL OF BIOCHEMISTRY AND CELL BIOLOGY, (1999 Oct) 31 (10) 1053-74. Ref: 171
Journal code: CDK. ISSN: 1357-2725.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 200004
EW 20000403
AB c-**Kit** is a receptor tyrosine kinase that binds **stem cell factor** (SCF). Structurally, c-**Kit** contains five immunoglobulin-like domains extracellularly and a catalytic domain divided into two regions by a 77 amino acid insert intracellularly. Studies in white spotting and steel mice have shown that functional SCF and c-**Kit** are critical in the survival and development of stem cells involved in hematopoiesis, pigmentation and reproduction. Mutations in c-**Kit** are associated with a variety of human diseases. Interaction of SCF with c-**Kit** rapidly induces receptor **dimerization** and increases in autophosphorylation activity. Downstream of c-**Kit**, multiple signal transduction components are activated, including phosphatidylinositol-3-kinase, Src family members, the JAK/STAT pathway and the Ras-Raf-MAP kinase cascade. Structure-function studies have begun to address the role of these signaling components in SCF-mediated responses. This review will focus on the biochemical mechanism of action of SCF in hematopoietic cells.
CT Check Tags: Animal; Human
ras Proteins: ME, metabolism
Dimerization
DNA-Binding Proteins: ME, metabolism
*Hematopoietic Stem Cells: ME, metabolism
Mice
Mitogen-Activated Protein Kinases: ME, metabolism
Phosphorylation
Protein-Tyrosine Kinase: ME, metabolism
***Proto-Oncogene Protein c-kit: ME, metabolism**
Proto-Oncogene Proteins c-raf: ME, metabolism
***Signal Transduction**
***Stem Cell Factor: ME, metabolism**
Structure-Activity Relationship
Trans-Activators: ME, metabolism
1-Phosphatidylinositol 3-Kinase: ME, metabolism
CN EC 2.7.1.- (Janus kinase 1); EC 2.7.1.- (Mitogen-Activated Protein Kinases); EC 2.7.1.112 (Protein-Tyrosine Kinase); EC 2.7.1.137 (1-Phosphatidylinositol 3-Kinase); EC 2.7.10.- (Proto-Oncogene Proteins c-raf); EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); EC 3.6.1.- (ras Proteins); 0 (gamma-activated factor, 91-kD); 0 (DNA-Binding Proteins); **0 (Stem Cell Factor)**; 0 (Trans-Activators)

L113 ANSWER 6 OF 47 MEDLINE

AN 2000047616 MEDLINE
DN 20047616
TI **Stem cell factor**/c-kit system in spermatogenesis.
AU Mauduit C; Hamamah S; Benahmed M
CS INSERM U407, Faculte de Medecine Lyon-Sud, Oullins, France.. mauduit@lsgrisn1.univ-lyon1.fr
SO HUMAN REPRODUCTION UPDATE, (1999 Sep-Oct) 5 (5) 535-45. Ref: 92
Journal code: CUH. ISSN: 1355-4786.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200003
EW 20000302
AB One of the major unresolved questions with male infertility is the identification of the molecular origin of a great majority of the spermatogenetic arrests currently diagnosed as idiopathic male infertility. During the past years, several families of regulating factors have been implicated in spermatogenesis defects observed essentially in animal models. Among these factors are signalling molecules, and particularly the **stem cell factor** (SCF)/c-kit system. The SCF and its receptor c-kit are an appropriate example to illustrate the role of signalling molecules in the physiology and pathology of spermatogenesis. The SCF/c-kit regulates primordial germ cell migration, proliferation and apoptosis during fetal gonadal development. The SCF/c-kit also regulates spermatogonia proliferation in the adult animal. In mutant mice, abnormalities of the SCF/c-kit gene expression, such as gene deletion, point mutation, alternative splicing defect, lead to different types of spermatogenesis alterations (e.g. decrease in primordial germ cell migration, decrease in spermatogonia proliferation). More recently, defects in SCF/c-kit gene expression have also been shown in human testicular dysfunctions. Indeed, a reduction in SCF/c-kit expression has been evidenced in oligozoospermia/azoospermia associated with an increase in the germ cell apoptosis process. In addition, c-kit seems to be a good marker of seminoma testicular tumours. This review reports a large number of data--obtained essentially in animal models--that suggest an important role for the SCF/c-kit system in spermatogenesis and, as a corollary, its potential involvement in spermatogenic defects.
CT Check Tags: Animal; Human; Male; Support, Non-U.S. Gov't
Apoptosis
Cell Division
Cell Movement
Gene Expression Regulation
***Infertility, Male: PP, physiopathology**
Mice
Mice, Mutant Strains
Proto-Oncogene Protein c-kit: GE, genetics
***Proto-Oncogene Protein c-kit: PH, physiology**
Rats
***Signal Transduction: PH, physiology**
***Spermatogenesis: PH, physiology**
Stem Cell Factor: GE, genetics
***Stem Cell Factor: PH, physiology**
Testis: CY, cytology
Testis: EM, embryology
Yolk Sac: CY, cytology
CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); **0 (Stem Cell Factor)**

L113 ANSWER 7 OF 47 MEDLINE
AN 1999455234 MEDLINE
DN 99455234
TI Isoforms of c-KIT differ in activation of signalling pathways and

transformation of NIH3T3 fibroblasts.
AU Caruana G; Cambareri A C; Ashman L K
CS Division of Haematology, Hanson Centre for Cancer Research, Institute of Medical and Veterinary Science, Adelaide, SA 5000, Australia.
SO ONCOGENE, (1999 Sep 30) 18 (40) 5573-81.
Journal code: ONC. ISSN: 0950-9232.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 200001
EW 20000104
AB Alternate splicing of mRNA encoding c-KIT results in isoforms which differ in the presence or absence of four amino acids (GNNK) in the juxtamembrane region of the extracellular domain of the receptor. In this study we show that these isoforms of human c-KIT, expressed at similar levels in NIH3T3 cells, display differential effects on various attributes of transformation. The GNNK- isoform strongly promoted anchorage independent growth (colony formation in semi-solid medium), loss of contact inhibition (focus formation), and led to tumorigenicity in nude mice. In contrast, the GNNK+ isoform elicited colony formation but relatively poor focus formation and no tumorigenicity. Saturation **binding** analysis indicated that the isoforms do not differ significantly in their affinity for the KIT **ligand**, Steel Factor (SLF). Negligible **ligand**-independent receptor phosphorylation was observed in either case but, after **ligand** stimulation, the GNNK- isoform displayed more rapid and extensive tyrosine autophosphorylation and faster internalization. Both isoforms recruited the p85 subunit of phosphatidylinositol 3-kinase and led to similar phosphorylation of its downstream effector c-Akt, but the GNNK- isoform gave rise to more MAP kinase phosphorylation. Thus the c-KIT isoforms display different signalling characteristics and have different transforming activity in NIH3T3 cells.
CT Check Tags: Animal; Comparative Study; Human; Support, Non-U.S. Gov't
Amino Acid Sequence
Cell Adhesion
*Cell Transformation, Neoplastic: ME, metabolism
DNA, Complementary: GE, genetics
Mice
Mice, Nude
Protein Isoforms: GE, genetics
*Protein Isoforms: PH, physiology
***Proto-Oncogene Protein c-kit: PH, physiology**
Recombinant Fusion Proteins: GE, genetics
Recombinant Fusion Proteins: PH, physiology
*RNA Splicing
***Signal Transduction: PH, physiology**
Stem Cell Factor: PH, physiology
Transfection
Tumor Stem Cell Assay
1-Phosphatidylinositol 3-Kinase: PH, physiology
3T3 Cells: PA, pathology
3T3 Cells: TR, transplantation
CN EC 2.7.1.137 (1-Phosphatidylinositol 3-Kinase); EC 2.7.11.- (Proto-Oncogene Protein c-kit); 0 (DNA, Complementary); 0 (Protein Isoforms); 0 (Recombinant Fusion Proteins); **0 (Stem Cell Factor)**

L113 ANSWER 8 OF 47 MEDLINE
AN 1999408738 MEDLINE
DN 99408738
TI Signaling via Src family kinases is required for normal internalization of the receptor c-Kit.
AU Broudy V C; Lin N L; Liles W C; Corey S J; O'Laughlin B; Mou S; Linnekin D
CS Divisions of Hematology, Department of Medicine, University of Washington, Seattle, WA, USA.. vcbroudy@u.washington.edu
NC DK44194 (NIDDK)

DK43719 (NIDDK)
CA31615 (NCI)
+
SO BLOOD, (1999 Sep 15) 94 (6) 1979-86.
Journal code: A8G. ISSN: 0006-4971.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199912
AB **Stem cell factor** (SCF) exerts its biological effects by **binding** to a specific receptor, the tyrosine kinase c-Kit, which is expressed on the cell surface. Although normal cellular trafficking of growth factor receptors may play a critical role in the modulation of receptor function, the mechanisms that regulate the distribution of c-Kit on the cell surface and the internalization of c-Kit have not been fully defined. We investigated whether signal transduction via Src family kinases is required for normal c-Kit trafficking. Treatment of the SCF-responsive human hematopoietic cell line MO7e with the inhibitor of Src family kinases PP1 blocked SCF-induced capping of c-Kit and internalization of c-Kit. c-Kit was able to associate with clathrin in the presence of PP1, suggesting that entry of c-Kit into clathrin-coated pits occurs independently of Src family kinases. SCF-induced internalization of c-Kit was also diminished in the D33-3 lymphoid cell line in which expression of Lyn kinase was disrupted by homologous recombination. These results indicate that Src family kinases play a role in **ligand**-induced trafficking of c-Kit.
CT Check Tags: Human; Support, U.S. Gov't, P.H.S.
src-Family Kinases: AI, antagonists & inhibitors
src-Family Kinases: GE, genetics
***src-Family Kinases: ME, metabolism**
Cell Membrane: PH, physiology
Chemotaxis: DE, drug effects
Chemotaxis: PH, physiology
Clathrin: ME, metabolism
*Coated Pits, Cell-Membrane: PH, physiology
Gene Expression Regulation, Enzymologic
Kinetics
Leukemia
Proto-Oncogene Protein c-kit: DE, drug effects
***Proto-Oncogene Protein c-kit: ME, metabolism**
Pyrazoles: PD, pharmacology
Pyrimidines: PD, pharmacology
Recombination, Genetic
***Signal Transduction: PH, physiology**
Stem Cell Factor: PD, pharmacology
***Stem Cell Factor: PH, physiology**
Tumor Cells, Cultured
CN EC 2.7.11.- (src-Family Kinases); EC 2.7.11.- (Proto-Oncogene Protein c-kit); 0 (Clathrin); 0 (Pyrazoles); 0 (Pyrimidines); **0 (Stem Cell Factor)**; 0 (4-amino-5-(4-methylphenyl)-7-(tert-butyl)pyrazolo(3,4-d)pyrimidine)

L113 ANSWER 9 OF 47 MEDLINE
AN 1999348687 MEDLINE
DN 99348687
TI SCF-KIT pathway in human epidermal melanocyte homeostasis [letter].
AU Longley B J; Carter E L
SO JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1999 Jul) 113 (1) 139-40.
Journal code: IHZ. ISSN: 0022-202X.
CY United States
DT Letter
LA English
FS Priority Journals; Cancer Journals
EM 199910
CT Check Tags: Human

Epidermis: CY, cytology
Epidermis: PH, physiology
*Homeostasis
Melanocytes: CY, cytology
***Melanocytes: PH, physiology**
***Proto-Oncogene Protein c-kit: PH, physiology**
Signal Transduction
***Stem Cell Factor: PH, physiology**
CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); **0 (Stem Cell Factor)**

L113 ANSWER 10 OF 47 MEDLINE
AN 1999317075 MEDLINE
DN 99317075
TI Defective expression of the SHP-1 phosphatase in polycythemia vera.
AU Wickrema A; Chen F; Namin F; Yi T; Ahmad S; Uddin S; Chen Y H; Feldman L; Stock W; Hoffman R; Platanias L C
CS Department of Medicine, University of Illinois at Chicago, USA.. awickrem@uic.edu
SO EXPERIMENTAL HEMATOLOGY, (1999 Jul) 27 (7) 1124-32.
Journal code: EPR. ISSN: 0301-472X.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199909
AB The SHP-1 phosphatase associates with the receptors for erythropoietin, **stem cell factor**, and interleukin-3, and negatively regulates the mitogenic signals generated during engagement by their respective **ligands**. The erythroid progenitors of patients with polycythemia vera are hypersensitive to the mitogenic effects of these growth factors despite the fact that the numbers and **binding** affinities for their receptors are not increased. To determine whether post-receptor signaling defects may account for growth factor-hypersensitivity in polycythemia vera, we determined the expression of SHP-1 in highly purified erythroid progenitors from polycythemia vera patients. Our data demonstrate that in approximately 60% of the patients, expression of SHP-1 in the colony forming unit-erythroid population is diminished. The decreased expression of the protein may result from a transcriptional defect, as suggested by the diminished SHP-1 mRNA expression in the erythroid progenitors of these patients. Studies to determine the level of maturation of polycythemia vera and normal cells indicated that there was no difference between the two at early colony forming unit-erythroid stage of differentiation although polycythemia vera cells showed retarded differentiation kinetics at late colony forming unit-erythroid stage of differentiation. Furthermore, SHP-1 expression in normal colony forming unit-erythroid demonstrated downregulation of mRNA and protein levels during terminal differentiation, suggesting that its function is required for growth control during the early stages of erythropoiesis. These results indicate an important role for SHP-1 in the regulation of normal human erythroid progenitors and suggest that defective expression of the protein may contribute to the pathogenesis of polycythemia vera.
CT Check Tags: Human; Support, Non-U.S. Gov't
Cell Differentiation
Colony-Forming Units Assay
Enzyme Induction
Erythroid Progenitor Cells: DE, drug effects
Erythroid Progenitor Cells: EN, enzymology
Erythroid Progenitor Cells: PA, pathology
Erythropoiesis: DE, drug effects
Erythropoiesis: PH, physiology
Erythropoietin: PD, pharmacology
Heme: BI, biosynthesis
Phosphorylation
*Polycythemia Vera: EN, enzymology
Polycythemia Vera: GE, genetics

Polycythemia Vera: PA, pathology
Protein Processing, Post-Translational
Protein-Tyrosine-Phosphatase: BI, biosynthesis
***Protein-Tyrosine-Phosphatase: DF, deficiency**
Protein-Tyrosine-Phosphatase: GE, genetics
Protein-Tyrosine-Phosphatase: PH, physiology
Proto-Oncogene Protein c-kit: ME, metabolism
Receptors, Erythropoietin: ME, metabolism
Receptors, Interleukin-3: ME, metabolism
RNA, Messenger: BI, biosynthesis
Signal Transduction
Transcription, Genetic
RN 11096-26-7 (Erythropoietin); 14875-96-8 (Heme)
CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); EC 3.1.3.- (SH protein-tyrosine phosphatase); EC 3.1.3.48 (Protein-Tyrosine-Phosphatase); 0 (Receptors, Erythropoietin); 0 (Receptors, Interleukin-3); 0 (RNA, Messenger)

L113 ANSWER 11 OF 47 MEDLINE
AN 1999287893 MEDLINE
DN 99287893
TI STAT protein recruitment and activation in c-**Kit** deletion mutants.
AU Brizzi M F; Dentelli P; Rosso A; Yarden Y; Pegoraro L
CS Department of Internal Medicine, University of Turin, Turin 10126, Italy.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Jun 11) 274 (24) 16965-72. Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199909
AB **Stem cell factor** (SCF) and its tyrosine kinase receptor, c-**Kit**, play a crucial role in regulating migration and proliferation of melanoblasts, germ cells, and hemopoietic cell progenitors by activating a number of intracellular signaling molecules. Here we report that SCF stimulation of myeloid cells or fibroblasts ectopically expressing c-**Kit** induces physical association with and tyrosine phosphorylation of three signal transducers and activators of transcription (STATs) as follows: STAT1alpha, STAT5A, and STAT5B. Other STAT proteins are not recruited upon SCF stimulation. Recruitment of STATs leads to their **dimerization**, nuclear translocation, and binding to specific promoter-responsive elements. Whereas STAT1alpha, possibly in the form of **homodimers**, binds to the sis-inducible DNA element, STAT5 proteins, either as STAT5A/STAT5B or STAT5/STAT1alpha **heterodimers**, bind to the prolactin-inducible element of the beta-casein promoter. The tyrosine kinase activity of **Kit** appears essential for STAT activation since a kinase-defective mutant lacking a kinase insert domain was inactive in STAT signaling. However, another mutant that lacked the carboxyl-terminal region retained STAT1alpha activation and nuclear translocation but was unable to fully activate STAT5 proteins, although it mediated their transient phosphorylation. These results indicate that different intracellular domains of c-**Kit** are involved in activation of the various STAT proteins.
CT Check Tags: Support, Non-U.S. Gov't
Binding Sites
Biological Transport
Bone Marrow Cells: CY, cytology
Bone Marrow Cells: ME, metabolism
Cell Compartmentation
Cell Nucleus: ME, metabolism
Dimerization
*DNA-Binding Proteins: ME, metabolism
*Mutation
Phosphorylation

Protein Binding
***Proto-Oncogene Protein c-kit: GE, genetics**
Response Elements
Sequence Deletion
Signal Transduction
***Stem Cell Factor: PD, pharmacology**
*Trans-Activators: ME, metabolism
*Transcription Factors: ME, metabolism
Tyrosine: ME, metabolism
RN 55520-40-6 (Tyrosine)
CN EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); 0 (gamma interferon activation factor); 0 (mammary gland-specific nuclear factor); 0 (DNA-Binding Proteins); **0 (Stem Cell Factor)**; 0 (Trans-Activators); 0 (Transcription Factors)

L113 ANSWER 12 OF 47 MEDLINE
AN 1999252441 MEDLINE
DN 99252441
TI **Stem cell factor**-induced airway hyperreactivity in allergic and normal mice.
AU Campbell E; Hogaboam C; Lincoln P; Lukacs N W
CS University of Michigan Medical School, Department of Pathology, Ann Arbor, Michigan 48109-0602, USA.
NC AI36302 (NIAID)
HL59178 (NHLBI)
SO AMERICAN JOURNAL OF PATHOLOGY, (1999 Apr) 154 (4) 1259-65.
Journal code: 3RS. ISSN: 0002-9440.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199908
EW 19990802
AB The induction of airway hyperreactivity during allergic responses involves multiple ill-defined mechanisms. Recently a role for **stem cell factor** (SCF) in the development of allergic eosinophilic airway inflammation has been identified. In the present study we demonstrate that SCF has a role in both the inflammatory response and airway hyperreactivity. Neutralization of SCF or examination of SCF-mutant mice, which were deficient in SCF and pulmonary mast cells, demonstrated significant alterations in the allergen-induced airway hyperreactive responses. The reduced hyperreactivity response was accompanied by a significant reduction in eosinophil accumulation. To examine the direct role of SCF on airway hyperreactivity, we administered SCF into the airways of normal mice via intratracheal injections and demonstrated a dose dependent increase in airway hyperreactivity at 4 hours that was maintained at 24 hours after administration. Instillation of SCF into SCF-deficient (mast cell deficient) mice demonstrated significantly lower increases in airway hyperreactivity compared with the littermate controls with normal mast cell numbers. These studies demonstrate that locally expressed SCF can induce changes in airway physiology via mast cell activation, verifying the role of SCF in allergic airway inflammation and hyperreactivity.
CT Check Tags: Animal; Female; Support, U.S. Gov't, P.H.S.
Airway Resistance: DE, drug effects
Antigens, Helminth: PD, pharmacology
Bronchial Provocation Tests
Dose-Response Relationship, Drug
Eosinophils: DE, drug effects
IgG: PD, pharmacology
Mast Cells: DE, drug effects
Mast Cells: IM, immunology
Methacholine Chloride: PD, pharmacology
Mice
Mice, Inbred CBA
Mice, Mutant Strains

***Respiratory Hypersensitivity: IM, immunology**
Stem Cell Factor: AI, antagonists & inhibitors
***Stem Cell Factor: IM, immunology**
***Stem Cell Factor: PD, pharmacology**
Time Factors
Trachea: DE, drug effects
*Trachea: IM, immunology
RN 62-51-1 (Methacholine Chloride)
CN 0 (Antigens, Helminth); 0 (IgG); **0 (Stem Cell Factor)**

L113 ANSWER 13 OF 47 MEDLINE
AN 1999208021 MEDLINE
DN 99208021
TI Hepatocyte growth factor/scatter factor-MET signaling in neural crest-derived melanocyte development.
AU Kos L; Aronzon A; Takayama H; Maina F; Ponzetto C; Merlino G; Pavan W
CS Laboratory for Genetic Disease Research, National Human Genome Research Institute, National Institutes of Health, Bethesda, Maryland 20892-4472, USA.
SO PIGMENT CELL RESEARCH, (1999 Feb) 12 (1) 13-21.
Journal code: PIG. ISSN: 0893-5785.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199908
AB The mechanisms governing development of neural crest-derived melanocytes, and how alterations in these pathways lead to hypopigmentation disorders, are not completely understood. Hepatocyte growth factor/scatter factor (HGF/SF) signaling through the tyrosine-kinase receptor, MET, is capable of promoting the proliferation, increasing the motility, and maintaining high tyrosinase activity and melanin synthesis of melanocytes in vitro. In addition, transgenic mice that ubiquitously overexpress HGF/SF demonstrate hyperpigmentation in the skin and leptomenigenes and develop melanomas. To investigate whether HGF/ SF-MET signaling is involved in the development of neural crest-derived melanocytes, transgenic embryos, ubiquitously overexpressing HGF/SF, were analyzed. In HGF/SF transgenic embryos, the distribution of melanoblasts along the characteristic migratory pathway was not affected. However, additional ectopically localized melanoblasts were also observed in the dorsal root ganglia and neural tube, as early as 11.5 days post coitus (p.c.). We utilized an in vitro neural crest culture assay to further explore the role of HGF/SF-MET signaling in neural crest development. HGF/SF added to neural crest cultures increased melanoblast number, permitted differentiation into pigmented melanocytes, promoted melanoblast survival, and could replace mast-cell growth factor/Steel factor (MGF) in explant cultures. To examine whether HGF/SF-MET signaling is required for the proper development of melanocytes, embryos with a targeted Met null mutation (Met-/-) were analysed. In Met-/- embryos, melanoblast number and location were not overtly affected up to 14 days p.c. These results demonstrate that HGF/SF-MET signaling influences, but is not required for, the initial development of neural crest-derived melanocytes in vivo and in vitro.
CT Check Tags: Animal; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Cell Differentiation: DE, drug effects
Cell Division
Cells, Cultured
Embryo: DE, drug effects
Gestational Age
Hepatocyte Growth Factor: GE, genetics
*Hepatocyte Growth Factor: ME, metabolism
Hepatocyte Growth Factor: PD, pharmacology
Melanocytes: DE, drug effects
***Melanocytes: PH, physiology**
Mice
Mice, Transgenic
*Neural Crest: CY, cytology

*Neural Crest: EM, embryology
Neural Crest: ME, metabolism
***Proto-Oncogene Protein c-met: ME, metabolism**
***Signal Transduction: PH, physiology**
Stem Cell Factor: ME, metabolism
Stem Cell Factor: PD, pharmacology
RN 67256-21-7 (Hepatocyte Growth Factor)
CN EC 2.7.11.- (Proto-Oncogene Protein c-met); **0 (Stem Cell Factor)**

L113 ANSWER 14 OF 47 MEDLINE
AN 1999027821 MEDLINE
DN 99027821
TI [Oocyte apoptosis: when, how, why?].
L'apoptose ovocytaire: quand, comment, pourquoi?.
AU Driancourt M A; Fair T; Reynaud K
CS INRA-URA CNRS 1291, Monnaie, France.
SO CONTRACEPTION, FERTILITE, SEXUALITE, (1998 Jul-Aug) 26 (7-8) 522-7.
Journal code: BUD. ISSN: 1165-1083.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
LA French
EM 199901
EW 19990104
AB The store of primordial follicles used for folliculogenesis is formed during oogenesis. Its size is the consequence of three processes: oogonia multiplication, time of meiosis initiation and extent of loss of germ cells (atretic oogonia, oocytes at the pachytene stage and newly formed primordial follicles). Apoptosis is causing this loss but its mechanisms are poorly documented. Both death signals (TNT alpha, Fas ligand) and survival signals (LIF, kit ligand) are present in the embryonic gonad. The apoptotic cascade then involves bclz, bax and caspases since knock out of these genes alters the store of primordial follicles. Apoptosis also exists within primordial follicles in adult ovaries and involves oocyte death. Its control has not been extensively studied.
CT Check Tags: Female; Human
Adult
Antigens, CD95: PH, physiology
*Apoptosis: PH, physiology
English Abstract
Mitosis
***Oocytes: PH, physiology**
***Oogenesis: PH, physiology**
Ovarian Follicle: PH, physiology
Signal Transduction
Stem Cell Factor: PH, physiology
Transcription Factors: PH, physiology
Tumor Necrosis Factor: PH, physiology
CN 0 (Antigens, CD95); **0 (Stem Cell Factor)**; 0 (Transcription Factors); 0 (Tumor Necrosis Factor)

L113 ANSWER 15 OF 47 MEDLINE
AN 1999025939 MEDLINE
DN 99025939
TI Growth and differentiation of human **stem cell factor**/erythropoietin-dependent erythroid progenitor cells in vitro.
AU Panzenbock B; Bartunek P; Mapara M Y; Zenke M
CS Max-Delbruck-Centre for Molecular Medicine, MDC, Berlin, Germany; and the Humboldt University Berlin, Virchow Klinikum, Robert-Rossle-Klinik, Berlin, Germany.
SO BLOOD, (1998 Nov 15) 92 (10) 3658-68.
Journal code: A8G. ISSN: 0006-4971.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals

EM 199902
EW 19990204
AB **Stem cell factor** (SCF) and erythropoietin (Epo) effectively support erythroid cell development in vivo and in vitro. We have studied here an SCF/Epo-dependent erythroid progenitor cell from cord blood that can be efficiently amplified in liquid culture to large cell numbers in the presence of SCF, Epo, insulin-like growth factor-1 (IGF-1), dexamethasone, and estrogen. Additionally, by changing the culture conditions and by administration of Epo plus insulin, such progenitor cells effectively undergo terminal differentiation in culture and thereby faithfully recapitulate erythroid cell differentiation in vitro. This SCF/Epo-dependent erythroid progenitor is also present in CD34(+) peripheral blood stem cells and human bone marrow and can be isolated, amplified, and differentiated in vitro under the same conditions. Thus, highly homogenous populations of SCF/Epo-dependent erythroid progenitors can be obtained in large cell numbers that are most suitable for further biochemical and molecular studies. We demonstrate that such cells express the recently identified adapter protein p62(dok) that is involved in signaling downstream of the c-kit/SCF receptor. Additionally, cells express the cyclin-dependent kinase (CDK) inhibitors p21(cip1) and p27(kip1) that are highly induced when cells differentiate. Thus, the in vitro system described allows the study of molecules and signaling pathways involved in proliferation or differentiation of human erythroid cells.
CT Check Tags: Comparative Study; Human
Blood Cells: CY, cytology
Blood Cells: DE, drug effects
Bone Marrow Cells: CY, cytology
Bone Marrow Cells: DE, drug effects
Cell Differentiation: DE, drug effects
Cell Division: DE, drug effects
Cells, Cultured
Cyclins: BI, biosynthesis
Cyclins: GE, genetics
Dexamethasone: PD, pharmacology
Enzyme Induction
*Erythroid Progenitor Cells: CY, cytology
Erythroid Progenitor Cells: DE, drug effects
*Erythropoiesis: DE, drug effects
Erythropoietin: PD, pharmacology
Estrogens: PD, pharmacology
Fetal Blood: CY, cytology
Insulin: PD, pharmacology
Insulin-Like Growth Factor I: PD, pharmacology
Microtubule-Associated Proteins: BI, biosynthesis
Microtubule-Associated Proteins: GE, genetics
Organ Specificity
Phosphoproteins: BI, biosynthesis
Phosphoproteins: GE, genetics
Signal Transduction
Stem Cell Factor: PD, pharmacology
RN 11061-68-0 (Insulin); 11096-26-7 (Erythropoietin); 147604-94-2 (KIP1 protein); 50-02-2 (Dexamethasone); 67763-96-6 (Insulin-Like Growth Factor I)
CN 0 (p62(dok) protein); 0 (Cip1 protein); 0 (Cyclins); 0 (Estrogens); 0 (Microtubule-Associated Proteins); 0 (Phosphoproteins); **0 (Stem Cell Factor)**

L113 ANSWER 16 OF 47 MEDLINE
AN 1999011376 MEDLINE
DN 99011376
TI Signaling events during male germ cell differentiation: bases and perspectives.
AU Berruti G
CS Dipartimento di Biologia, Universit`a di Milano, via Celoria 26, 20133 Milano, Italy.

SO FRONTIERS IN BIOSCIENCE, (1998 Nov 1) 3 D1097-108. Ref: 117
Journal code: CUE. ISSN: 1093-4715.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199901
AB In all species, reproductive function depends on the ability of the individual to produce functional differentiated gametes. Spermatogenesis is a cyclic process in which diploid spermatogonia differentiate into mature haploid spermatozoa. Thus from a genetic point of view, spermatogenesis can be divided into two phases, namely the diploid and haploid phase. Indeed, this complex differentiation process is still more intriguing since primary spermatocytes, if genetically diploid, are functionally tetraploid, while elongating spermatids, the germ cells undergoing the most dramatic morphological changes, if genetically haploid, become functionally anucleate due to ongoing condensation of chromatin resulting in an inactive nuclear DNA. This multi-step differentiative pathway is dependent on a specific environment provided by the anatomical and cellular relationships that take place in the testis and more specifically within the seminiferous tubules. Already, early anatomists (mind comes to Enrico Sertoli and Gustaf Retzius) were fascinated by the mixed cellular composition of the testis correctly deciphered as a whole of interacting and interdependent cell types despite the fact these belong to two well-established and different cell lineages, i.e, the somatic and germinal line. Since their time (the XIX century) up to-day a conspicuous bulk of experimental work and a relative massive bibliographic documentation have been provided. From this it stands out : a) a sophisticated role played by the cyclic hormonal control elicited by the hypothalamic-pituitary axis; b) the structural membrane specializations of Sertoli-germ cell communications; c) the existence and action of a paracrine and autocrine testicular regulative secretion; d) a regulation of germ cell gene expression, highly specialized both at transcriptional, posttranscriptional, and translational level; e) an active participation of the haploid genome in the final steps of cell differentiation. Each of these points has been the matter of several more and less recent reviews to which the present author hands back in the course of this note. However all these points, although topics of separate and extensive treatises, are conceptually jointed by a 'leit-motiv', that is, the intracellular transduction of an exogenous signal evoking a specific stimulatory/inhibitory, proliferative/differentiative event. The spirit with which the present author interpreted this minireview was to recall some points to which to draw attention having as a scenario the complex process of male germ cell differentiation in mammals.
CT Check Tags: Animal; Human; Male; Support, Non-U.S. Gov't
*Cell Differentiation
DNA-Binding Proteins: PH, physiology
Estrogens: PH, physiology
Heat-Shock Proteins 70: PH, physiology
Models, Biological
Progesterone: PH, physiology
Proto-Oncogene Protein c-kit: PH, physiology
Receptors, Estrogen: PH, physiology
Receptors, Progesterone: PH, physiology
***Signal Transduction**
***Spermatozoa: PH, physiology**
Stem Cell Factor: PH, physiology
Testis: PH, physiology
RN 135844-64-3 (CREM protein); 57-83-0 (Progesterone)
CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); 0 (heat shock protein 70.2); 0 (DNA-Binding Proteins); 0 (Estrogens); 0 (Heat-Shock Proteins 70); 0 (Receptors, Estrogen); 0 (Receptors, Progesterone); **0 (Stem Cell Factor)**

L113 ANSWER 17 OF 47 MEDLINE
AN 1999002668 MEDLINE
DN 99002668
TI Lck associates with and is activated by Kit in a small cell lung cancer cell line: **inhibition** of SCF-mediated growth by the Src family kinase **inhibitor** PP1.
AU Krystal G W; DeBerry C S; Linnekin D; Litz J
CS Department of Medicine, Medical College of Virginia Commonwealth University, McGuire Veterans Affairs Medical Center, Richmond 23249, USA.. GKRYSTAL@GEMS.VCU.edu
SO CANCER RESEARCH, (1998 Oct 15) 58 (20) 4660-6.
Journal code: CNF. ISSN: 0008-5472.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199901
AB At least 70% of small cell lung cancers (SCLCs) express the Kit receptor tyrosine kinase and its ligand, **stem cell factor** (SCF). In an effort to define the signal transduction pathways activated by Kit in SCLC, we focused on Src family kinases and, in particular, Lck, a Src-related tyrosine kinase that is expressed in hemopoietic cells and certain tumors, including SCLC. SCF treatment of the H526 cell line induced a physical association between Kit and Lck that, in vitro, was dependent on phosphorylation of the juxtamembrane domain of Kit. Stimulation of Kit with recombinant SCF resulted in a rapid 3-6-fold increase in the specific activity of Lck, which was similar in magnitude to the activation of Lck resulting from the cross-linking of the T-cell receptor complex of Jurkat cells. Lck activity peaked by 5 min after SCF addition, and the elevated activity persisted for at least 30 min in the presence of SCF, with kinetics similar to the activation of mitogen-activated protein kinase. PP1, an **inhibitor** of Src family kinases with selectivity for Lck, completely **inhibited** SCF-mediated growth but had little effect on insulin-like growth factor-I-mediated growth. PP1 **antagonized** both SCF-mediated proliferation and **inhibition** of apoptosis. PP1 had no effect on Kit kinase activity but was shown to **block** total Lck activity by at least 90% by immune complex kinase assay. Low levels of Src, Hck, and Yes were also expressed in the H526 cell line; only Yes showed a consistent increase in specific activity, which was also **inhibited** by PP1 following SCF treatment. These data demonstrate that, in the H526 SCLC cell line, Lck and, possibly, Yes are downstream of Kit in a signal transduction pathway; the **inhibition** by PP1 of SCF-mediated proliferation and **inhibition** of apoptosis suggests that Src family kinases are intermediates in the signaling pathways that regulate these processes.
CT Check Tags: Human; Support, U.S. Gov't, Non-P.H.S.
***src-Family Kinases: AI, antagonists & inhibitors**
***Carcinoma, Small Cell: DT, drug therapy**
Carcinoma, Small Cell: ME, metabolism
***Enzyme Inhibitors: PD, pharmacology**
Jurkat Cells
*Lung Neoplasms: DT, drug therapy
Lung Neoplasms: ME, metabolism
***Lymphocyte Specific Protein Tyrosine Kinase p56(lck): PH, physiology**
***Proto-Oncogene Protein c-kit: PH, physiology**
Pyrazoles: PD, pharmacology
Pyrimidines: PD, pharmacology
Signal Transduction
***Stem Cell Factor: AI, antagonists & inhibitors**
Stem Cell Factor: PD, pharmacology
CN EC 2.7.11.- (src-Family Kinases); EC 2.7.11.- (Lymphocyte Specific Protein Tyrosine Kinase p56(lck)); EC 2.7.11.- (Proto-Oncogene Protein c-kit); 0 (Enzyme **Inhibitors**); 0 (Pyrazoles); 0 (Pyrimidines); **0 (Stem Cell Factor)**; 0 (4-amino-5-(4-methylphenyl)-7-(tert-

butyl)pyrazolo(3,4-d)pyrimidine)

L113 ANSWER 18 OF 47 MEDLINE
AN 1998430685 MEDLINE
DN 98430685
TI **Stem cell factor** augments Fc epsilon RI-mediated TNF-alpha production and stimulates MAP kinases via a different pathway in MC/9 mast cells.
AU Ishizuka T; Kawasome H; Terada N; Takeda K; Gerwins P; Keller G M; Johnson G L; Gelfand E W
CS Department of Pediatrics, National Jewish Medical and Research Center, Denver, CO 80206, USA.
NC AI HL-36577 (NIAID)
AI 4224B (NIAID)
DK-37871 (NIDDK)
SO JOURNAL OF IMMUNOLOGY, (1998 Oct 1) 161 (7) 3624-30.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199812
AB Mast cells express the receptor tyrosine kinase **kit/stem cell factor** receptor (SCFR) which is encoded by the proto-oncogene c-**kit**. Ligation of SCFR induces its **dimerization** and activation of its intrinsic tyrosine kinase activity leading to activation of Raf-1, phospholipases, phosphatidylinositol 3-kinase, and extracellular signal-regulated kinases. However, little is known about the downstream signals initiated by SCFR ligation except for activation of extracellular signal-regulated kinases. The murine mast cell line, MC/9, synthesizes and secretes TNF-alpha following the aggregation of high affinity Fc receptors for IgE (Fc epsilonRI). Ligation of SCFR or Fc epsilonRI on MC/9 cells resulted in the activation of all three MAP kinase family members, extracellular signal-regulated kinases, c-Jun amino-terminal kinase (JNK), and p38. **Stem cell factor** (SCF)-induced activation of JNK and p38 was insensitive to wortmannin, cyclosporin A, and FK506 whereas activation of these kinases through Fc epsilonRI was sensitive to these drugs. Coligation of SCFR augmented Fc epsilonRI-mediated activation of MAP kinases, especially JNK activation, and SCF augmented Fc epsilonRI-mediated TNF-alpha production in MC/9 cells, although SCF alone did not induce TNF-alpha production. This augmentation by SCF was regulated at the level of transcription, at least in part, since the promoter activity of TNF-alpha was enhanced following addition of SCF. These results demonstrate that SCF can augment Fc epsilonRI-mediated JNK activation and cytokine gene transcription but via pathways that are regulated differently than the ones activated through Fc epsilonRI.
CT Check Tags: Animal; Support, U.S. Gov't, P.H.S.
*Adjuvants, Immunologic: PH, physiology
Amino Acid Sequence
Androstadienes: PD, pharmacology
Antigens: PD, pharmacology
Ca(2+)-Calmodulin Dependent Protein Kinase: DE, drug effects
***Ca(2+)-Calmodulin Dependent Protein Kinase: ME, metabolism**
Cell Line
Cyclosporine: PD, pharmacology
Enzyme Activation: DE, drug effects
Enzyme Activation: IM, immunology
Gene Expression Regulation: IM, immunology
*Mast Cells: EN, enzymology
Mast Cells: IM, immunology
Mast Cells: ME, metabolism
Mice
Molecular Sequence Data
Ovalbumin: IM, immunology
Ovalbumin: PD, pharmacology

Polyenes: PD, pharmacology
Promoter Regions (Genetics): IM, immunology
Protein-Serine-Threonine Kinases: AI, antagonists & inhibitors
Protein-Serine-Threonine Kinases: ME, metabolism
Proto-Oncogene Protein c-kit: ME, metabolism
Receptors, IgE: DE, drug effects
Receptors, IgE: ME, metabolism
*Receptors, IgE: PH, physiology
Signal Transduction: IM, immunology
Stem Cell Factor: DE, drug effects
Stem Cell Factor: ME, metabolism
***Stem Cell Factor: PH, physiology**
Tacrolimus: PD, pharmacology
*Tumor Necrosis Factor: BI, biosynthesis
Tumor Necrosis Factor: GE, genetics

RN 109581-93-3 (Tacrolimus); 19545-26-7 (wortmannin); 53123-88-9 (Sirolimus); 59865-13-3 (Cyclosporine); 9006-59-1 (Ovalbumin)

CN EC 2.7.10 (Protein-Serine-Threonine Kinases); EC 2.7.10.- (c-Jun amino-terminal kinase); EC 2.7.10.- (mitogen-activated protein kinase p38); EC 2.7.10.- (p42 MAP Kinase); EC 2.7.10.- (AKT1 protein kinase); EC 2.7.10.- (Ca(2+)-Calmodulin Dependent Protein Kinase); EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); 0 (Adjuvants, Immunologic); 0 (Androstadienes); 0 (Antigens); 0 (Polyenes); 0 (Receptors, IgE); **0 (Stem Cell Factor)**; 0 (Tumor Necrosis Factor)

L113 ANSWER 19 OF 47 MEDLINE

AN 1998413782 MEDLINE

DN 98413782

TI Morphological alterations in rat peritoneal mast cells by **stem cell factor**.

AU Kim H M; Shin H Y; Lee E H

CS Department of Oriental Pharmacy, College of Pharmacy, Wonkwang University, Iksan, Chonbuk, South Korea.

SO IMMUNOLOGY, (1998 Jun) 94 (2) 242-6.
Journal code: GH7. ISSN: 0019-2805.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Cancer Journals

EM 199812

EW 19981202

AB **Stem cell factor** (SCF) stimulates mast cell adhesion and, because SCF is produced normally in tissues, it may be a major factor responsible for the adhesion of mast cells to connective tissue matrix. We found that the morphology of rat peritoneal mast cells (RPMC) altered after the addition of recombinant murine SCF (rmSCF) in vitro. The ability of rmSCF to enhance morphological alteration was dose dependent and completely abolished by anti-c-kit **ACK2** monoclonal antibody. Exposure of RPMC to transforming growth factor-beta 1, wortmannin, genistein, herbimycin A, staurosporine, indomethacin and cytochalasin D before the addition of rmSCF **antagonized** rmSCF-induced morphological alteration. However, nordihydroguiaretic acid had no effect. Many RPMC appeared to respond also to nerve growth factor (NGF) but the total number of cells with altered morphology was much greater when the culture was stimulated by rmSCF than by NGF. We suggest that morphological alterations of mast cells by rmSCF is an important step for the participation in adhesion to tissue under resident physiological conditions.

CT Check Tags: Animal; Support, Non-U.S. Gov't
Cell Culture
Dose-Response Relationship, Drug
*Mast Cells: CY, cytology
Nerve Growth Factors: PD, pharmacology
*Peritoneal Cavity: CY, cytology
Rats

Rats, Wistar
Recombinant Proteins: PD, pharmacology
Stem Cell Factor: AI, antagonists & inhibitors
***Stem Cell Factor: PD, pharmacology**
Transforming Growth Factor beta: PD, pharmacology
CN 0 (Nerve Growth Factors); 0 (Recombinant Proteins); **0 (Stem Cell Factor)**; 0 (Transforming Growth Factor beta)

L113 ANSWER 20 OF 47 MEDLINE
AN 1998324991 MEDLINE
DN 98324991
TI Lineage-specific signaling in melanocytes. C-**kit** stimulation recruits p300/CBP to microphthalmia.
AU Price E R; Ding H F; Badalian T; Bhattacharya S; Takemoto C; Yao T P; Hemesath T J; Fisher D E
CS Pediatric Hematology/Oncology, Dana Farber Cancer Research Institute and Harvard Medical School, Boston, Massachusetts 02115, USA.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1998 Jul 17) 273 (29) 17983-6. Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199810
AB During melanocyte development, the cytokine Steel factor activates its receptor c-**Kit**, initiating a signal transduction cascade, which is vital for lineage determination via unknown downstream nuclear targets. c-**Kit** has recently been found to trigger mitogen-activated protein kinase-mediated phosphorylation of Microphthalmia (Mi), a lineage-restricted transcription factor, which, like Steel factor and c-**Kit**, is essential for melanocyte development. This cascade results in increased Mi-dependent transcriptional reporter activity. Here we examine the mechanism by which Mi is activated by this pathway. Phosphorylation does not significantly alter Mi's nuclear localization, DNA binding, or **dimerization**. However, the transcriptional coactivator p300/CBP selectively associates with mitogen-activated protein kinase-phosphorylated Mi, even under conditions in which non-MAPK phospho-Mi is more abundant. Moreover, p300/CBP coactivates Mi transcriptional activity in a manner dependent upon this phosphorylation. Mi thus joins CREB as a transcription factor whose signal-responsive phosphorylation regulates coactivator recruitment, in this case modulating lineage development in melanocytes.
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Ca(2+)-Calmodulin Dependent Protein Kinase: ME, metabolism
Dimerization
DNA-Binding Proteins: GE, genetics
*DNA-Binding Proteins: PH, physiology
Enzyme Activation
Hamsters
***Melanocytes: PH, physiology**
Mice
*Nuclear Proteins: PH, physiology
Phosphorylation
Protein Binding
Proto-Oncogene Protein c-kit: PH, physiology
Rabbits
***Signal Transduction**
Stem Cell Factor: PH, physiology
Trans-Activation (Genetics)
*Trans-Activators: PH, physiology
*Transcription Factors: PH, physiology
Tumor Cells, Cultured
CN EC 2.7.10.- (Ca(2+)-Calmodulin Dependent Protein Kinase); EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); 0 (CREB-binding protein); 0 (DNA-Binding Proteins); 0 (E1A-associated

p300 protein); 0 (Mi protein); 0 (Nuclear Proteins); 0 (**Stem Cell Factor**); 0 (Trans-Activators); 0 (Transcription Factors)

L113 ANSWER 21 OF 47 MEDLINE
AN 1998234426 MEDLINE
DN 98234426
TI Role of **dimerization** of the membrane-associated growth factor **kit** ligand in juxtacrine signaling: the Sl17H mutation affects **dimerization** and stability-phenotypes in hematopoiesis.
AU Tajima Y; Huang E J; Vosseller K; Ono M; Moore M A; Besmer P
CS Molecular Biology Program, Memorial Sloan-Kettering Cancer Center, New York, New York 10021, USA.
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1998 May 4) 187 (9) 1451-61. Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199808
EW 19980801
AB The **Kit** ligand (KL)/**Kit** receptor pair functions in hematopoiesis, gametogenesis, and melanogenesis. KL is encoded at the murine steel (Sl) locus and encodes a membrane growth factor which may be proteolytically processed to produce soluble KL. The membrane-associated form of KL is critical in mediating **Kit** function in vivo. Evidence for a role of cytoplasmic domain sequences of KL comes from the Sl17H mutation, a splice site mutation that replaces the cytoplasmic domain with extraneous amino acids. Using deletion mutants and the Sl17H allele, we have investigated the role of the cytoplasmic domain sequences of KL in biosynthetic processing and cell surface presentation. The normal KL protein products are processed for cell surface expression, where they form **dimers**. Both Sl17H and the cytoplasmic deletion mutants of KL were processed to the cell surface; however, the rate of transport and protein stability were affected by the mutations. Deletion of cytoplasmic domain sequences of KL did not affect **dimerization** of KL. In contrast, **dimerization** of the Sl17H protein was reduced substantially. In addition, we have characterized the hematopoietic cell compartment in Sl17H mutant mice. The Sl17H mutation has only minor effects on hematopoiesis. Tissue and peritoneal mast cell numbers were reduced in mutant mice as well as in myeloid progenitors. Interestingly, long-term bone marrow cultures from Sl17H mice did not sustain the long-term production of hematopoietic cells. In addition, homing of normal hematopoietic progenitors to the spleen of irradiated Sl17H/Sl17H recipient mice was diminished in transplantation experiments, providing evidence for a role of **Kit** in homing or lodging. These results demonstrate that the membrane forms of KL exist as **homodimers** on the cell surface and that **dimerization** may play an important role in KL/**Kit**-mediated juxtacrine signaling.
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Amino Acid Sequence
Bone Marrow Cells: ME, metabolism
COS Cells
Dimerization
Flow Cytometry
Hematopoiesis: GE, genetics
*Hematopoiesis: PH, physiology
Mast Cells: ME, metabolism
Mice
Microscopy, Fluorescence
Molecular Sequence Data
Mutation: GE, genetics
RNA Splicing: GE, genetics
Sequence Deletion: GE, genetics
Signal Transduction: PH, physiology
***Stem Cell Factor: CH, chemistry**

Stem Cell Factor: PH, physiology
Stem Cells: ME, metabolism
CN **0 (Stem Cell Factor)**

L113 ANSWER 22 OF 47 MEDLINE
AN 1998152084 MEDLINE
DN 98152084
TI The c-kit receptor and its possible signaling transduction pathway in mouse spermatozoa.
AU Feng H; Sandlow J I; Sandra A
CS Department of Urology, University of Iowa, Iowa City 52242-1089, USA.
SO MOLECULAR REPRODUCTION AND DEVELOPMENT, (1998 Mar) 49 (3) 317-26. Journal code: AN7. ISSN: 1040-452X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199806
AB The presence and role of the c-kit protein was investigated in the mature sperm of the mouse. The c-kit monoclonal antibody (mAb) **ACK2** reacted specifically with the acrosomal region and the principal piece of fixed noncapacitated sperm but did not react with the acrosome region in acrosome-reacted sperm. **ACK2** significantly inhibited the acrosome reaction; this inhibition was relieved by the calcium ionophore A23187. The kit **ligand stem cell factor** (SCF) significantly increased the percentage of sperm undergoing acrosome reaction. This increase was partially inhibited by the calcium channel inhibitor (verapamil), the PI3k inhibitor (wortmannin), and the PLC inhibitor (U-73122). **ACK2** predominantly recognized c-kit proteins of 33, 48, and 150 kDa by Western blotting of mouse sperm extracts. The 48- and 150-kDa protein bands were released into the media and tyrosine autophosphorylated at low basal levels during acrosome reaction. On stimulation with SCF, the level of c-kit phosphorylation increased significantly. These findings suggest that c-kit is present in mature sperm, and its **binding** to SCF may result in the activation of PLC gamma 1 and PI3K, leading to receptor autophosphorylation, and ultimately may play a role in capacitation and/or the acrosome reaction.
CT Check Tags: Animal; Male; Support, Non-U.S. Gov't
Antibodies, Monoclonal: PD, pharmacology
Blotting, Western
Enzyme Inhibitors: PD, pharmacology
Immunoenzyme Techniques
Isoenzymes: ME, metabolism
Mice
Phospholipase C: ME, metabolism
Phosphorylation
Protein Kinase C: ME, metabolism
***Proto-Oncogene Protein c-kit: ME, metabolism**
Rabbits
Rats
***Signal Transduction**
Spermatozoa: DE, drug effects
***Spermatozoa: ME, metabolism**
Stem Cell Factor: PD, pharmacology
Tyrosine: ME, metabolism
1-Phosphatidylinositol 3-Kinase: ME, metabolism
RN 55520-40-6 (Tyrosine)
CN EC 2.7.1.- (Protein Kinase C); EC 2.7.1.137 (1-Phosphatidylinositol 3-Kinase); EC 2.7.11.- (Proto-Oncogene Protein c-kit); EC 3.1.4.- (phospholipase C gamma); EC 3.1.4.3 (Phospholipase C); 0 (Antibodies, Monoclonal); 0 (Enzyme Inhibitors); 0 (Isoenzymes); **0 (Stem Cell Factor)**

L113 ANSWER 23 OF 47 MEDLINE
AN 1998070948 MEDLINE

DN 98070948
TI Activation of the receptor tyrosine kinase Kit is required for the proliferation of melanoblasts in the mouse embryo.
AU Mackenzie M A; Jordan S A; Budd P S; Jackson I J
CS MRC Human Genetics Unit, Western General Hospital, Edinburgh, EH4 2XU, United Kingdom.
SO DEVELOPMENTAL BIOLOGY, (1997 Dec 1) 192 (1) 99-107.
Journal code: E7T. ISSN: 0012-1606.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199803
AB The development of neural crest-derived melanocytes, as well as haematopoietic and germ cells, is affected by mutations of the Kit and Mgf genes, which lead to dominant spotting (W) or steel (Sl) phenotypes. Mgf codes for the ligand of the receptor tyrosine kinase encoded by the Kit locus. KitW-v, a point mutation exerting a dominant negative effect, causes a substantial reduction in tyrosine kinase activity of the Kit receptor and leads to a characteristic pigmentation phenotype, namely dilute coat colour and a white ventral and head spot with reduced pigmentation of the feet and tail in the heterozygous animal, as well as slight anaemia. Homozygous animals lack coat pigmentation and are severely anaemic and infertile. Dct is a marker for cells of the melanoblast lineage. In order to study these cells in detail we have generated transgenic mouse lines carrying the lacZ reporter under the control of the Dct promoter and have used the embryonic expression of the reporter to identify early melanoblasts before they begin to produce pigment. Our transgenic lines have simplified the study of melanoblasts in the mouse embryo, and by crossing our mice with KitW-v mutants we have been able to identify the midgestation stages at which melanoblasts rely critically on Mgf/Kit interactions. We conclude that the survival of immature melanoblasts depends crucially upon Kit signalling up until E11, and later in development Kit plays a vital role in melanoblast proliferation. Our data do not describe a dependence upon Kit for melanoblast migration or differentiation. Copyright 1997 Academic Press.
CT Check Tags: Animal; Female; Male; Support, Non-U.S. Gov't
Base Sequence
Cell Differentiation: GE, genetics
Cell Division: GE, genetics
Cell Movement: GE, genetics
Crosses, Genetic
DNA Primers: GE, genetics
Enzyme Activation
Gene Expression Regulation, Developmental
Genetic Markers
Lac Operon
***Melanocytes: CY, cytology**
***Melanocytes: EN, enzymology**
Mice
Mice, Transgenic
Phenotype
Pigmentation Disorders: EM, embryology
Pigmentation Disorders: GE, genetics
Point Mutation
Proto-Oncogene Protein c-kit: GE, genetics
***Proto-Oncogene Protein c-kit: ME, metabolism**
Signal Transduction: GE, genetics
Stem Cell Factor: GE, genetics
CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); 0 (DNA Primers); 0 (Genetic Markers); **0 (Stem Cell Factor)**

L113 ANSWER 24 OF 47 MEDLINE
AN 1998008115 MEDLINE
DN 98008115
TI Signal transduction in human hematopoietic cells by vascular endothelial

growth factor related protein, a novel ligand for the FLT4 receptor.
AU Wang J F; Ganju R K; Liu Z Y; Avraham H; Avraham S; Groopman J E
CS Division of Experimental Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA 02115, USA.
NC HL 53745-02 (NHLBI)
HL 43510-07 (NHLBI)
HL 55187-01 (NHLBI)
+
SO BLOOD, (1997 Nov 1) 90 (9) 3507-15.
Journal code: A8G. ISSN: 0006-4971.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199801
AB We have recently identified a novel **ligand of** the vascular endothelial growth factor (VEGF) family termed VEGF-related protein (VRP), which specifically **binds** to the FLT4 receptor. To characterize the signaling events after VRP engagement of its cognate receptor in hematopoietic cells, a population of human erythroleukemia (HEL) cells, termed HEL-JW, expressing high levels of FLT4 receptor was isolated. Stimulation of HEL-JW cells with VRP alone and in combination with the c-kit **ligand/stem cell factor** increased cell growth. VRP induced tyrosine phosphorylation of various proteins, including the FLT4 receptor. Further characterization of these tyrosine phosphorylated molecules revealed that Shc, Grb2, and SOS form a complex with the activated FLT4 receptor. HEL-JW cells also expressed RAFTK, a recently identified member of the focal adhesion kinase family. RAFTK was phosphorylated and activated upon VRP treatment, and there was an enhanced association of this kinase with the adaptor protein Grb2. Furthermore, the c-Jun NH2-terminal kinase (JNK), involved in growth activation and shown to mediate RAFTK signaling in other cell types, was activated by VRP stimulation. We also observed that VRP treatment of HEL-JW cells resulted in the phosphorylation of the cytoskeletal protein paxillin. This treatment resulted in an increased association of paxillin with RAFTK, which was mediated by the C-terminal region of RAFTK. These studies indicate that VRP stimulation induced the formation of a signaling complex at its activated receptor as well as activation of RAFTK. VRP-mediated activation of RAFTK may facilitate signal transduction to the cytoskeleton and downstream to the JNK pathway in FLT4-expressing blood cells.
CT Check Tags: Human; Support, U.S. Gov't, P.H.S.
*Carrier Proteins: PH, physiology
Cells, Cultured
*Hematopoietic Stem Cells: PH, physiology
Ligands
***Receptor Protein-Tyrosine Kinases: PH, physiology**
*Receptors, Cell Surface: PH, physiology
***Signal Transduction**
RN 144638-77-7 (FLT4 protein)
CN EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Carrier Proteins); 0 (Ligands); 0 (Receptors, Cell Surface); 0 (VEGF-related protein)

L113 ANSWER 25 OF 47 MEDLINE
AN 97359263 MEDLINE
DN 97359263
TI A new strategy for treating small cell lung cancer.
AU Ueda R; Takashi T
CS Department of Internal Medicine II, Nagoya City University Medical School.
SO NIHON KYOBU SHIKKAN GAKKAI ZASSHI. JAPANESE JOURNAL OF THORACIC DISEASES, (1996 Dec) 34 Suppl 111-4. Ref: 7
Journal code: KQD. ISSN: 0301-1542.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA Japanese
EM 199711
AB Recent results from molecular biology have shown that lung cancer is characterized by multiple, sequentially appearing molecular changes that include genetic and epigenetic alterations. Among all types of lung cancer, small cell lung cancer (SCLC) is associated with the lowest rate of 5-year survival. In this symposium, we introduce our findings regarding the c-kit oncogenes in SCLC. We found that the c-kit gene is strongly expressed in SCLC. The c-kit gene was not expressed in normal bronchial epithelial cells, which indicates that this gene is abberantly transcribed in SCLC. In addition, c-kit-positive cases of SCLC showed autophosphorylation in response to recombinant human **stem cell factor**. Furthermore, adding rh **stem cell factor** of SCLC cell lines induced a significant chemotactic response and moderate in vitro cell growth. These results strongly suggest that abnormal expression of the c-kit gene may be involved in the pathogenesis of SCLC by autocrine/paracrine stimulation via the c-kit/SCF **signal pathway**. To overcome drug resistance, we assessed the efficacy of a chimeric toxin targeted to c-kit receptors.
CT Check Tags: Human
Carcinoma, Small Cell: GE, genetics
***Carcinoma, Small Cell: TH, therapy**
English Abstract
Gene Expression Regulation, Neoplastic
*Immunotoxins: TU, therapeutic use
Lung Neoplasms: GE, genetics
***Lung Neoplasms: TH, therapy**
Proto-Oncogene Protein c-kit: GE, genetics
Stem Cell Factor: ME, metabolism
CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); 0 (Immunotoxins); **0 (Stem Cell Factor)**

L113 ANSWER 26 OF 47 MEDLINE
AN 97334241 MEDLINE
DN 97334241
TI The IL-4 receptor alpha-chain cytoplasmic domain is sufficient for activation of JAK-1 and STAT6 and the induction of IL-4-specific gene expression.
AU Reichel M; Nelson B H; Greenberg P D; Rothman P B
CS Department of Dermatology, Columbia University College of Physicians and Surgeons, New York, NY 10032, USA.
NC AI33540-04 (NIAID)
AI36613 (NIAID)
CA18029 (NCI)
SO JOURNAL OF IMMUNOLOGY, (1997 Jun 15) 158 (12) 5860-7.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199709
AB The common gamma-chain (gamma(c)) is a functional component of the IL-4R, yet cells lacking gamma(c) are able to respond to IL-4. This has led to the suggestion that a surrogate gamma'-chain, which can interact with the IL-4R alpha chain to mediate signaling, is expressed on cells lacking gamma(c). An alternative possibility is that in the absence of gamma(c), the IL-4R alpha chain is able to transduce signals by **homodimerization**. To test this latter possibility, a chimeric receptor containing the extracellular domain of c-**kit** (the **stem cell factor** (SCF) receptor) and the cytoplasmic and transmembrane domains of the IL-4R alpha chain was generated. Treatment of cells expressing the chimeric receptor **kit** /IL-4R alpha with SCF induces activation of the IL-4R alpha-associated kinase JAK-1 and the transcription factor STAT6. However, tyrosine phosphorylation of JAK-3, which associates with gamma(c), is not induced

by SCF in these cells. SCF-mediated ligation of **kit**/IL-4R alpha is sufficient to elicit IL-4-specific gene **expression**, including up-regulation of CD23 and synthesis of germ-line epsilon transcripts. In the T cell line CTLL2, ligation of **kit**/IL-4R alpha induces cellular proliferation. Finally, in JAK-1-deficient HeLa cells, STAT6 activation by IL-4 is completely abolished. Together, these data demonstrate that the IL-4R alpha cytoplasmic domain is sufficient to activate JAK-1 and STAT6 and to induce expression of IL-4 target genes, thus identifying a mechanism by which IL-4 signaling can proceed in the absence of JAK-3 and gamma(c).

CT Check Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
*Antigens, CD: AN, analysis
Base Sequence
Cells, Cultured
Enzyme Activation
*Gene Expression
*Interleukin-4
Lymphocyte Transformation
Molecular Sequence Data
Phosphorylation
***Protein-Tyrosine Kinase: ME, metabolism**
Proto-Oncogene Protein c-kit: AN, analysis
*Receptors, Interleukin: AN, analysis
***Signal Transduction**
T-Lymphocytes: IM, immunology
*Trans-Activators: ME, metabolism
Tyrosine: ME, metabolism

RN 168115-60-4 (Stat6 protein); 55520-40-6 (Tyrosine)

CN EC 2.7.1.- (Janus kinase 1); EC 2.7.1.- (Janus kinase 3); EC 2.7.1.112 (Protein-Tyrosine Kinase); EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); 0 (Antigens, CD); 0 (Interleukin-4); 0 (Receptors, Interleukin); 0 (Receptors, Interleukin-4); 0 (Trans-Activators)

L113 ANSWER 27 OF 47 MEDLINE

AN 97329531 MEDLINE

DN 97329531

TI Cross-linking of integrins induces tyrosine phosphorylation of the proto-oncogene product Vav and the protein tyrosine kinase Syk in human factor-dependent myeloid cells.

AU Gotoh A; Takahira H; Geahlen R L; Broxmeyer H E

CS Department of Microbiology and Immunology, Indiana University, School of Medicine, Indianapolis 46202-5121, USA.

NC R01 HL56416 (NHLBI)
R01 HL54037 (NHLBI)
P01 HL53586 (NHLBI)
+

SO CELL GROWTH AND DIFFERENTIATION, (1997 Jun) 8 (6) 721-9.
Journal code: AYH. ISSN: 1044-9523.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199709

AB Attachment to extracellular matrix is important in the regulation of proliferation and differentiation of hematopoietic stem and progenitor cells. Post-**ligand** occupancy events of integrin receptors in myeloid cells are largely unknown. We examined early signaling events after stimulation of integrin receptors (outside-in signal) using a cross-linking system in a growth factor-dependent myeloid cell line, M07e, alpha 4, alpha 5, and beta 1 integrin cross-linking induced a similar pattern of transient tyrosine phosphorylation of cellular proteins. The approximate molecular weights of these phosphoproteins were M(r) 150,000, M(r) 120,000-125,000, M(r) 95,000, M(r) 70,000, M(r) 60,000, and M(r) 40,000-50,000. Vav, Syk, and Erk2 were identified as some of the tyrosine-phosphorylated proteins, and their weights were M(r) 95,000, M(r)

70,000, and M(r) 40,000-50,000, respectively. Erk2 and Vav were also tyrosine-phosphorylated by stimulation with Steel factor (SLF) and granulocyte macrophage colony-stimulating factor, whereas tyrosine phosphorylation of Syk was not induced by stimulation with these cytokines. The degree of tyrosine phosphorylation of Vav through integrin engagement was almost equal to that by SLF stimulation, whereas that of Erk2 was much weaker than with SLF stimulation. Upon integrin engagement, antibodies raised against Syk coprecipitated several tyrosine-phosphorylated proteins. In vitro **binding** assays demonstrated that, among these Syk-associated proteins, pp40, which differed from Erks, Crk, and Crkl, **binds** Syk through SH2 domains of Syk and is a prominent tyrosine-phosphorylated protein in integrin cross-linked cells. These results suggest that tyrosine phosphorylation of Vav and Erk2 in myeloid cells might be regulated by both integrins and cytokines in the bone marrow microenvironment, whereas Syk might be involved in a distinct pathway from the shared between integrins and cytokines in myeloid cells.

CT Check Tags: Human; Support, U.S. Gov't, P.H.S.
Blotting, Western
Ca(2+)-Calmodulin Dependent Protein Kinase: ME, metabolism
Cell Adhesion
Cell Line
Cytokines: ME, metabolism
***Enzyme Precursors: ME, metabolism**
Growth Substances: ME, metabolism
*Hematopoietic Stem Cells: ME, metabolism
*Integrins: ME, metabolism
Phosphorylation
***Protein-Tyrosine Kinase: ME, metabolism**
*Proto-Oncogene Proteins: ME, metabolism
Receptors, Fibronectin: ME, metabolism
Recombinant Fusion Proteins
Signal Transduction
Stem Cell Factor: ME, metabolism
Tyrosine: ME, metabolism

RN 55520-40-6 (Tyrosine)

CN EC 2.7.1.- (myelin basic protein kinase); EC 2.7.1.- (p72syk); EC 2.7.1.112 (Protein-Tyrosine Kinase); EC 2.7.10.- (Ca(2+)-Calmodulin Dependent Protein Kinase); 0 (proto-oncogene protein vav); 0 (Cytokines); 0 (Enzyme Precursors); 0 (Growth Substances); 0 (Integrins); 0 (Proto-Oncogene Proteins); 0 (Receptors, Fibronectin); 0 (Recombinant Fusion Proteins); **0 (Stem Cell Factor)**

L113 ANSWER 28 OF 47 MEDLINE

AN 97303716 MEDLINE

DN 97303716

TI Cytokines involved in B-cell differentiation and their sites of action.

AU Takatsu K

CS Department of Immunology, University of Tokyo, Japan.

SO PROCEEDINGS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE, (1997 Jun) 215 (2) 121-33. Ref: 171
Journal code: PXZ. ISSN: 0037-9727.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)

LA English

FS Priority Journals; Cancer Journals

EM 199708

AB B cells originate from pluripotent hematopoietic stem cells and differentiate in the bone marrow into mature B cells. The differentiation of a stem cell into a mature B cell can be subdivided into five steps: early pro-B cells, late pro-B cell stage, pre-B cell stage, immature B cells, and mature B cells. Each differentiation step appears to be regulated by co-receptor and cytokines. The earliest B-cell progenitors are bound to the stromal cell surface by adhesive interactions through cell surface molecules to promote the **binding** of c-kit to

stem cell factor (SCF). At the late pro-B cell stage, interleukin-7 (IL-7) induces proliferation and differentiation of pro-B cells to pre-B cells. Surface Ig-expressing mature B cells leave bone marrow and circulate into peripheral lymphoid organs in which they can be activated to proliferate and to differentiate into antibody-secreting cells by encountering antigens and "helper" T (TH) cells. TH cells activate B cells by their products, cytokines such as IL-4, IL-5, and IL-6, and membrane-bound stimulatory molecules including CD40 **ligand**. Each cytokine has pleiotropic activity on B cells and other cell types, and acts through a specific receptor. Abnormal expression of a cytokine receptor and aberrant signal transduction causes functional abnormality of B cells.

CT Check Tags: Animal; Human
Antigens, CD: PH, physiology
Antigens, Differentiation: PH, physiology
*B-Lymphocytes: CY, cytology
Cell Differentiation
Cell Division
*Cytokines: PH, physiology
Interleukin-4: PH, physiology
Interleukin-5: PH, physiology
Interleukin-6: PH, physiology
Lymphocyte Transformation
Nucleosidases: PH, physiology
Receptors, Antigen, B-Cell: PH, physiology
Receptors, Interleukin: PH, physiology
Signal Transduction
T-Lymphocytes, Helper-Inducer: PH, physiology

CN EC 3.2.2. (Nucleosidases); EC 3.2.2.- (T10 antigen); 0 (Antigens, CD); 0 (Antigens, Differentiation); 0 (Cytokines); 0 (Interleukin-4); 0 (Interleukin-5); 0 (Interleukin-6); 0 (Receptors, Antigen, B-Cell); 0 (Receptors, Interleukin); 0 (Receptors, Interleukin-6)

L113 ANSWER 29 OF 47 MEDLINE
AN 96290410 MEDLINE
DN 96290410
TI Interaction of **stem cell factor** and its receptor c-kit mediates lodgment and acute expansion of hematopoietic cells in the murine spleen.
AU Broudy V C; Lin N L; Priestley G V; Nocka K; Wolf N S
CS Department of Medicine University of Washington, Seattle 98195, USA.
NC DK44194 (NIDDK)
AG07724 (NIA)
SO BLOOD, (1996 Jul 1) 88 (1) 75-81.
Journal code: A8G. ISSN: 0006-4971.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199611
AB The phenotypes of mice that harbor a defect in the genes encoding either **stem cell factor** (SCF) or its receptor, c-kit, indicate that this ligand/receptor pair is necessary for maintenance of normal hematopoiesis in the adult. Our objective was to determine whether SCF, like erythropoietin, is necessary for acute erythroid expansion during recovery from hemolytic anemia. Monoclonal antibody **ACK2**, which recognizes the murine c-kit receptor, was used to selectively **block** the hematopoietic growth-promoting effects of SCF. Mice were treated with phenylhydrazine on day 0 and day 1 to induce hemolytic anemia and also received no antibody, control IgG, or **ACK2** on day 0. The mice were killed on day 3 and the hematocrit (Hct), reticulocyte count, and numbers of erythroid and myeloid hematopoietic progenitor cells (colony-forming unit-erythroid [CFU-E], burst-forming unit [BFU]-E, and CFU-granulocyte-macrophage [GM]) were quantitated in the femoral marrow and spleen using hematopoietic colony-forming assays. Induction of hemolytic anemia with phenylhydrazine resulted in a drop in the Hct from

approximately 50% to 30%, and an approximate 8- to 10-fold increase in the reticulocyte count. The numbers of CFU-E increased modestly in the femur, and approximately 25- to 50-fold in the spleen, in comparison with normal mice. BFU-E and CFU-GM values did not increase in the femur but expanded 6- to 10-fold in the spleen, in comparison with normal mice. This confirms that much of the erythroid expansion in response to hemolytic anemia occurs in the murine spleen. Neutralizing quantities of the **ACK2** antibody reduced femoral CFU-E, BFU-E, and CFU-GM content to less than half that found in phenylhydrazine-treated control mice and nearly totally ablated splenic hematopoiesis. These results suggest that c-kit receptor function may be required for optimal response to acute erythropoietic demand and that erythropoiesis in the splenic microenvironment is more dependent on SCF/c-kit receptor interaction than is erythropoiesis in the marrow microenvironment. Because expansion of late erythropoiesis in the spleen was preferentially **blocked**, we tested the hypothesis that homing of more primitive hematopoietic cells to the spleen was dependent on c-kit receptor function. Lethally irradiated mice were injected with marrow cells obtained from mice that had received phenylhydrazine plus control IgG or with marrow cells obtained from mice that had received phenylhydrazine plus **ACK2**. In parallel experiments, normal murine marrow cells were treated in vitro with control IgG or with **ACK2** and were injected into lethally irradiated mice. The fraction of BFU-E and CFU-GM retrieved from the marrow and spleen of the recipient mice 4 hours later was reduced by approximately 75% when progenitor cells had been exposed to **ACK2**, in comparison with control IgG. These data suggest that interaction of SCF with the c-kit receptor affects the homing behavior of hematopoietic progenitor cells in the adult animal.

CT Check Tags: Animal; Comparative Study; Female; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Anemia, Hemolytic: CI, chemically induced
Anemia, Hemolytic: PA, pathology
Antibodies, Monoclonal: PD, pharmacology
Bone Marrow: PA, pathology
Cell Movement: PH, physiology
Colony-Forming Units Assay
Erythropoiesis: PH, physiology
Hematopoietic Stem Cells: ME, metabolism
*Hematopoietic Stem Cells: PA, pathology
IgG: PD, pharmacology
Mice
Mice, Inbred C57BL
Mice, Inbred DBA
Phenotype
Phenylhydrazines: TO, toxicity
Proto-Oncogene Protein c-kit: DE, drug effects
***Proto-Oncogene Protein c-kit: PH, physiology**
Radiation Chimera
Rats
*Spleen: PA, pathology
Stem Cell Factor: AI, antagonists & inhibitors
***Stem Cell Factor: PH, physiology**

RN 100-63-0 (phenylhydrazine)

CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); 0 (Antibodies, Monoclonal); 0 (IgG); 0 (Phenylhydrazines); **0 (Stem Cell Factor)**

L113 ANSWER 30 OF 47 MEDLINE

AN 96202494 MEDLINE

DN 96202494

TI The RAR-RXR as well as the RXR-RXR pathway is involved in signaling growth inhibition of human CD34+ erythroid progenitor cells.

AU Rusten L S; Dybedal I; Blomhoff H K; Blomhoff R; Smeland E B; Jacobsen S E

CS Department of Immunology, Institute for Cancer Research, The Norweigian Radium Hospital, Oslo.

SO BLOOD, (1996 Mar 1) 87 (5) 1728-36.
Journal code: A8G. ISSN: 0006-4971.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 199609
AB Previous studies have shown that retinoic acid (RA), similar to tumor necrosis factor-alpha (TNF-alpha), can act as a bifunctional regulator of the growth of bone marrow progenitors, in that it can stimulate granulocyte-macrophage colony-stimulating factor (GM-CSF)- or interleukin-3 (IL-3)-induced GM colony formation, but potently inhibit G-CSF-induced growth. The present study, using highly enriched human CD34+ as well as Lin- murine bone marrow progenitor cells, demonstrates a potent inhibitory effect of 9-cis-RA on burst-forming unit-erythroid (BFU-E) colony formation regardless of the cytokine stimulating growth. Specifically, 9-cis-RA potently inhibited the growth of BFU-E response to erythropoietin (Epo) (100%), **stem cell factor** (SCF) + Epo (92%), IL-3 + Epo (97%), IL-4 + Epo (88%), and IL-9 + Epo (100%). Erythroid colony growth was also inhibited when CD34+ progenitors were seeded at one cell per well, suggesting a direct action of RA. Using synthetic **ligands** to retinoic acid receptors (RARs) and retinoid X receptors (RXRs) that selectively **bind** and activate RAR-RXR or RXR-RXR **dimers**, respectively, we dissected the involvement of the two retinoid response pathways in the regulation of normal myeloid and erythroid progenitor cell growth. Transactivation studies showed that both the RAR (Ro 13-7410) and RXR (Ro 25-6603 and Ro 25-7386) **ligands** were highly selective at 100 nmol/L. At this concentration, Ro 13-7410 potently inhibited G-CSF-stimulated myeloid as well as SCF + Epo-induced erythroid colony growth. At the same concentration, Ro 25-6603 and Ro 25-7386 had little or no effect on G-CSF-induced colony formation, whereas they inhibited 75% and 53%, respectively, of SCF + Epo-stimulated BFU-E colony growth. Thus, the RAR-RXR response pathway can signal growth inhibition of normal bone marrow myeloid and erythroid progenitor cells. In addition, we demonstrate a unique involvement of the RXR-RXR pathway in mediating growth inhibition of erythroid but not myeloid progenitor cells.
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't
Antigens, CD34
Base Sequence
Benzoates: PD, pharmacology
Consensus Sequence
Cyclohexanes: PD, pharmacology
Depression, Chemical
*Erythroid Progenitor Cells: CY, cytology
Erythroid Progenitor Cells: DE, drug effects
*Erythropoiesis: DE, drug effects
Erythropoietin: PD, pharmacology
Hematopoietic Cell Growth Factors: PD, pharmacology
Interleukins: PD, pharmacology
Mice
Mice, Inbred BALB C
Molecular Sequence Data
Pentanoic Acids: PD, pharmacology
Rats
Receptors, Retinoic Acid: AG, agonists
Receptors, Retinoic Acid: DE, drug effects
*Receptors, Retinoic Acid: PH, physiology
Recombinant Proteins: PD, pharmacology
Retinoids: PD, pharmacology
***Signal Transduction: PH, physiology**
Stem Cell Factor: PD, pharmacology
Transcription Factors: DE, drug effects
*Transcription Factors: PH, physiology
Tretinoin: PD, pharmacology
RN 11096-26-7 (Erythropoietin); 302-79-4 (Tretinoin); 71441-28-6 (Ro 13-7410)
CN 0 (retinoic acid receptor alpha); 0 (retinoid X receptor); 0 (Antigens, CD34); 0 (Benzoates); 0 (Cyclohexanes); 0 (Hematopoietic Cell Growth Factors); 0 (Interleukins); 0 (Pentanoic Acids); 0 (Receptors, Retinoic Acid); 0 (Recombinant Proteins); 0 (Retinoids); 0 (Ro 25-6603); 0

(Stem Cell Factor); 0 (Transcription Factors)

L113 ANSWER 31 OF 47 MEDLINE
AN 96195144. MEDLINE
DN 96195144
TI Signalling mechanisms of endothelin-induced mitogenesis and melanogenesis in human melanocytes.
AU Imokawa G; Yada Y; Kimura M
CS Institute for Fundamental Research, Kao Corporation, Tochigi, Japan.
SO BIOCHEMICAL JOURNAL, (1996 Feb 15) 314 (Pt 1) 305-12.
Journal code: 9YO. ISSN: 0264-6021.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Cancer Journals; Priority Journals
EM 199610
AB To understand the signalling mechanisms involved in the dual stimulatory effects of endothelin-1 (ET-1) on DNA synthesis and melanization in cultured human melanocytes, we analysed the biological profile of ET-1 receptor and determined the effects of ET-1 on the protein kinase C, cyclic AMP system and mitogen-activated protein kinase (MAP kinase) in comparison with their relevant stimulants. The photoaffinity labelling of ET-1 receptors with Denny-Jaff reagents revealed an ET-1 receptor with a molecular mass of 51 kDa in human melanocytes. The ET(A) receptor subtype-sensitive antagonist BQ123(50 nM) or pertussis toxin (100 ng/ml) significantly suppressed the ET-1-induced intracellular calcium mobilization, indicating the presence of pertussis toxin-sensitive G-protein-coupled ET(A) receptors. An assay of protein kinase C activity revealed that 10nM ET-1 translocated cytosolic protein kinase C to membrane-bound protein kinase C within 5 min of the start of incubation. In contrast, receptor-mediated melanocyte activation by ET-1 was accompanied by an elevated level of cyclic AMP (4-fold over control) after 10-60 min of incubation, whereas 60 min of incubation of human melanocytes with c-Kit or c-Met ligands such as **stem cell factor** (10 nM) or basic fibroblast growth factor (10 nM) did not elevate the cyclic AMP level. We have also demonstrated that a specific tyrosine kinase inhibitor, tyrphostin B-42 (10 microM), inhibited the ET-1-induced growth stimulation, suggesting the involvement of the tyrosine kinase pathway in growth stimulation. Consistently, an assay of MAP kinase revealed that ET-1 caused a 10-fold activation of MAP kinase after 5 min of incubation with human melanocytes in a similar way to tyrosine kinase ligands such as **stem cell factor** and hepatocyte growth factor. Further, the DNA synthesis stimulated by the c-Kit ligand **stem cell factor** at a concentration of 1 nM was synergistically enhanced by 5 nM ET-1. These results suggest that ET-induced dual cellular events in human melanocytes are closely associated with cross-talk between the protein kinase C and A and tyrosine kinase pathways.
CT Check Tags: Human
Amino Acid Sequence
Ca(2+)-Calmodulin Dependent Protein Kinase: ME, metabolism
Cholera Toxin: PD, pharmacology
Cyclic AMP: ME, metabolism
DNA: BI, biosynthesis
DNA: DE, drug effects
*Endothelins: PD, pharmacology
***Melanins: BI, biosynthesis**
Melanocytes: CY, cytology
***Melanocytes: ME, metabolism**
Molecular Sequence Data
Peptides, Cyclic: PD, pharmacology
Pertussis Toxins: PD, pharmacology
Phosphodiesterase Inhibitors: PD, pharmacology
Protein Kinase C: AI, antagonists & inhibitors
Protein Kinase C: ME, metabolism
Protein-Tyrosine Kinase: ME, metabolism

Receptors, Endothelin: AI, antagonists & inhibitors
Receptors, Endothelin: CH, chemistry
*Receptors, Endothelin: ME, metabolism
*Signal Transduction
Stem Cell Factor: PD, pharmacology
Thiouracil: ME, metabolism
1-Methyl-3-isobutylxanthine: PD, pharmacology
RN 136553-81-6 (BQ 123); 141-90-2 (Thiouracil); 28822-58-4 (1-Methyl-3-isobutylxanthine); 60-92-4 (Cyclic AMP); 70323-44-3 (Pertussis Toxins); 9007-49-2 (DNA); 9012-63-9 (Cholera Toxin)
CN EC 2.7.1.- (Protein Kinase C); EC 2.7.1.112 (Protein-Tyrosine Kinase); EC 2.7.10.- (extracellular signal-regulated kinase 1); EC 2.7.10.- (p42 MAP Kinase); EC 2.7.10.- (Ca(2+)-Calmodulin Dependent Protein Kinase); 0 (endothelin A receptor); 0 (Endothelins); 0 (Melanins); 0 (Peptides, Cyclic); 0 (Phosphodiesterase Inhibitors); 0 (Receptors, Endothelin); 0 (Stem Cell Factor)

L113 ANSWER 32 OF 47 MEDLINE
AN 96019555 MEDLINE
DN 96019555
TI Biology of flt3 ligand and receptor.
AU Lyman S D
CS Immunex Corporation, Seattle, WA 98101, USA.
SO INTERNATIONAL JOURNAL OF HEMATOLOGY, (1995 Aug) 62 (2) 63-73. Ref: 42
Journal code: A7F. ISSN: 0925-5710.
CY Ireland
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
EM 199606
AB The flt3 ligand is a member of a small family of growth factors that stimulate the proliferation of hematopoietic cells; other members of this family include Steel factor (also known as mast cell growth factor, stem cell factor, and kit ligand) and colony stimulating factor 1. These proteins function by binding to and activating unique tyrosine kinase receptors. Expression of the flt3 receptor is primarily restricted among hematopoietic cells to the most primitive progenitor cells. The flt3 ligand is similar to Steel factor in that both proteins stimulate the proliferation of early progenitor or stem cells. Neither of these factors has much proliferative activity on its own, but each factor can synergize with a wide range of other colony stimulating factors and interleukins (ILs) to stimulate proliferation. One major difference between the two factors appears to be their effect on mast cells, which Steel factor stimulates but flt3 ligand does not. Although flt3 ligand and Steel factor each act on early hematopoietic cells, differences in their activities suggest that they are not redundant and both are required for normal hematopoiesis. There are a number of clinical settings in which the flt3 ligand may potentially prove quite useful.
CT Check Tags: Animal; Human
Amino Acid Sequence
Cell Line
Cloning, Molecular
*Hematopoietic Stem Cells: PH, physiology
Leukemia: GE, genetics
Leukemia: PA, pathology
Liver: CY, cytology
Liver: EM, embryology
*Membrane Proteins: PH, physiology
Membrane Proteins: TU, therapeutic use
Mice
Molecular Sequence Data
Multigene Family
Phosphorylation

Protein Conformation
Protein Processing, Post-Translational
Proto-Oncogene Protein c-kit: PH, physiology
*Proto-Oncogene Proteins: PH, physiology
***Receptor Protein-Tyrosine Kinases: PH, physiology**
Receptor, Macrophage Colony-Stimulating Factor: PH, physiology
Signal Transduction
Stem Cell Factor: PH, physiology
Tumor Cells, Cultured

CN EC 2.7.1.- (fetal liver kinase-2); EC 2.7.11.- (Proto-Oncogene Protein c-kit); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); EC 2.7.11.- (Receptor, Macrophage Colony-Stimulating Factor); 0 (flt3 ligand protein); 0 (Membrane Proteins); 0 (Proto-Oncogene Proteins); **0 (Stem Cell Factor)**

L113 ANSWER 33 OF 47 MEDLINE
AN 95294029 MEDLINE
DN 95294029
TI Identification of the major phosphorylation sites for protein kinase C in **kit/stem cell factor** receptor in vitro and in intact cells.
AU Blume-Jensen P; Wernstedt C; Heldin C H; Ronnstrand L
CS Ludwig Institute for Cancer Research, Uppsala Branch, Biomedical Center, Sweden.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Jun 9) 270 (23) 14192-200.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199509
AB The c-**kit**-encoded tyrosine kinase receptor for **stem cell factor** (**Kit**/SCFR) is crucial for the development of hematopoietic cells, melanoblasts, and germ cells. Ligand stimulation of **Kit**/SCFR leads to receptor **dimerization** and autophosphorylation on tyrosine residues. We recently showed, that protein kinase C (PKC) acts in an SCF-stimulated negative feedback loop, which controls **Kit**/SCFR tyrosine kinase activity and modulates the cellular responses to SCF (Blume-Jensen, P., Siegbahn, A., Stabel, S., Heldin, C.-H., and Ronnstrand, L. (1993) EMBO J. 12, 4199-4209). We present here the identification of the major phosphorylation sites for PKC in **Kit**/SCFR. Two serine residues in the kinase insert, Ser-741 and Ser-746, are PKC-dependent phosphorylation sites in vivo and account for all phosphorylation by PKC in vitro. Together they comprise more than 60% of the total SCF-stimulated receptor phosphorylation in living cells and 85-90% of its phosphorylation in resting cells. Two additional serine residues, Ser-821 close to the major tyrosine autophosphorylation site in the kinase domain and Ser-959 in the carboxyl terminus are SCF-stimulated PKC-dependent phosphorylation sites. However, they are not phosphorylated directly by PKC-alpha in vitro. Both specific receptor tyrosine autophosphorylation and specific receptor-associated phosphatidylinositide 3'-kinase activity was increased approximately 2-fold in response to SCF in PAE cells stably expressing **Kit**/SCFR(S741A/S746A). Furthermore, the kinase activity of **Kit**/SCFR(S741A/S746A) toward an exogenous substrate was increased, which was reflected as a decreased Km and an increased Vmax, in accordance with the negative regulatory role of PKC on **Kit**/SCFR signaling.
CT Check Tags: Support, Non-U.S. Gov't
Base Sequence
Hematopoietic Cell Growth Factors: PD, pharmacology
Molecular Sequence Data
Phosphorylation
Phosphotransferases (Alcohol Group Acceptor): ME, metabolism
Polycyclic Hydrocarbons: PD, pharmacology
***Protein Kinase C: PH, physiology**
*Proto-Oncogene Proteins: ME, metabolism

*Receptor Protein-Tyrosine Kinases: ME, metabolism
*Receptors, Colony-Stimulating Factor: ME, metabolism
Signal Transduction
Tetradecanoylphorbol Acetate: PD, pharmacology
Transfection

RN 121263-19-2 (calphostin C); 16561-29-8 (Tetradecanoylphorbol Acetate)

CN EC 2.7.1 (Phosphotransferases (Alcohol Group Acceptor)); EC 2.7.1.- (Protein Kinase C); EC 2.7.1.137 (1-Phosphatidylinositol 3-Kinase); EC 2.7.11.- (Proto-Oncogene Protein c-kit); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Hematopoietic Cell Growth Factors); 0 (Polycyclic Hydrocarbons); 0 (Proto-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); 0 (Stem Cell Factor)

L113 ANSWER 34 OF 47 MEDLINE

AN 95211033 MEDLINE

DN 95211033

TI The flt3 ligand: a hematopoietic stem cell factor whose activities are distinct from steel factor.

AU Lyman S D; Brasel K; Rousseau A M; Williams D E

CS Immunex Research and Development Corporation, Seattle, Washington.

SO STEM CELLS, (1994) 12 Suppl 1 99-107; discussion 108-10. Ref: 28
Journal code: BN2. ISSN: 1066-5099.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 199507

AB A number of growth factors have been described that affect the hematopoietic system. Among this group are Steel factor (also known as mast cell growth factor, stem cell factor and kit ligand), and the more recently described flt3 ligand. These factors have been shown to function by binding to and activating the c-kit and flt3 tyrosine kinase receptors, respectively. Both of these factors stimulate the growth of mouse and human hematopoietic progenitor cells. These factors therefore differ from such later acting hematopoietic factors as colony-stimulating factor (CSF)-1, which regulates the growth, survival and differentiation of monocytic cells through the c-fms tyrosine kinase receptor. Like Steel factor, the flt3 ligand has little biological activity on its own, but synergizes well with a number of other colony stimulating factors and interleukins. One major difference between the two factors appears to be their effect on mast cells. Steel factor stimulates both the proliferation and activation of mast cells, while preliminary data with the flt3 ligand suggests that it has no effect on mast cells. Although the flt3 ligand and Steel factor each act on early hematopoietic cells, differences in their activities suggest that they are not redundant and are both required for normal hematopoiesis.

CT Check Tags: Animal; Female; Human; Male
Cloning, Molecular
Gene Expression
*Hematopoiesis: PH, physiology
Hematopoietic Cell Growth Factors: CH, chemistry
Hematopoietic Cell Growth Factors: GE, genetics
*Hematopoietic Cell Growth Factors: PH, physiology
Hematopoietic Stem Cells: CY, cytology
Leukemia: PP, physiopathology
Mast Cells: CY, cytology
Membrane Proteins: CH, chemistry
Membrane Proteins: GE, genetics
*Membrane Proteins: PH, physiology
Molecular Structure
Proto-Oncogene Proteins: CH, chemistry
Proto-Oncogene Proteins: GE, genetics

Proto-Oncogene Proteins: PH, physiology
Receptor Protein-Tyrosine Kinases: CH, chemistry
Receptor Protein-Tyrosine Kinases: GE, genetics
Receptor Protein-Tyrosine Kinases: PH, physiology
Signal Transduction
CN EC 2.7.1.- (fetal liver kinase-2); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (flt3 ligand protein); 0 (Hematopoietic Cell Growth Factors); 0 (Membrane Proteins); 0 (Proto-Oncogene Proteins); **0 (Stem Cell Factor)**

L113 ANSWER 35 OF 47 MEDLINE
AN 95151825 MEDLINE
DN 95151825
TI Steel factor and c-**kit** protooncogene: genetic lessons in signal transduction.
AU Lev S; Blechman J M; Givol D; Yarden Y
CS Department of Chemical Immunology, Weizmann Institute of Science, Rehovot, Israel.
SO CRITICAL REVIEWS IN ONCOGENESIS, (1994) 5 (2-3) 141-68. Ref: 166
Journal code: A1Y. ISSN: 0893-9675.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199505
AB Despite extensive research on the molecular mechanisms of signal transduction by growth factors and their oncogenic receptor tyrosine kinases, the physiological relevance of these pathways, especially in mammals, remains largely unknown. A unique exception is the Steel factor (SLF) and its c-**kit**-encoded receptor, because many natural germ line mutations of both the ligand and the receptor exist in mice. The protooncogene c-**kit** encodes a cell surface receptor that belongs to the immunoglobulin gene family and carries an intrinsic tyrosine kinase activity in its cytoplasmic portion. The precursor of the **Kit** ligand, SLF, is also a transmembrane protein that exists as a soluble factor as well as a cell surface protein. The interaction of **Kit** with SLF leads to receptor **dimerization**, kinase activation, and tyrosine phosphorylation of cytoplasmic proteins that contain Src homology 2 motifs. Various mutations in **Kit** and SLF result in a defective signaling pathway and underly the complex phenotypes of W and Sl mice, respectively. The early development of at least four cell lineages is affected. These are erythrocytes, melanocytes, germ cells, and mast cells. Correlation between the behavior of these lineages and specific mutations uncovered interesting physiological aspects of the mechanism of signal transduction by a polypeptide growth factor. These include the different degrees of severity of affected lineages, indications for distinct functions during early embryonic development and at late phases, the significance of synergy between a growth factor and lymphokines, the interaction between mutant and wild-type proteins in heterozygous animals, and the possibility that a surface-anchored ligand may act differently than a soluble factor. Predictably, the lessons learned with **Kit** and Sl mice will be widely relevant to other pairs of ligands and receptors that control the function of different cell lineages and physiological processes.
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
*Hematopoietic Cell Growth Factors: GE, genetics
Mice
*Mutation
*Proto-Oncogene Proteins: GE, genetics
***Receptor Protein-Tyrosine Kinases: GE, genetics**
*Receptors, Colony-Stimulating Factor: GE, genetics
***Signal Transduction: GE, genetics**
CN EC 2.7.11.- (**Proto-Oncogene Protein**

c-kit); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Hematopoietic Cell Growth Factors); 0 (**Proto**-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); **0 (Stem Cell Factor)**

L113 ANSWER 36 OF 47 MEDLINE
AN 95112336 MEDLINE
DN 95112336
TI The fourth immunoglobulin domain of the **stem cell factor** receptor couples **ligand binding** to signal transduction.
AU Blechman J M; Lev S; Barg J; Eisenstein M; **Vaks** B; Vogel Z; Givol D; Yarden Y
CS Department of Chemical Immunology, Weizmann **Institute** of Science, Rehovot, Israel.
SO CELL, (1995 Jan 13) 80 (1) 103-13.
Journal code: CQ4. ISSN: 0092-8674.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199504
AB Receptor **dimerization** is ubiquitous to the **action** of all receptor tyrosine kinases, and in the case **of dimeric ligands**, such as the **stem cell factor** (SCF), it was attributed to **ligand** bivalency. However, by using a **dimerization**-inhibitory monoclonal antibody to the SCF receptor, we confined a putative **dimerization** site to the nonstandard fourth immunoglobulin-like domain of the receptor. Deletion of this domain not only abolished **ligand**-induced **dimerization** and completely inhibited signal transduction, but also provided insights into the mechanism of the coupling of **ligand binding** to **dimer** formation. These results identify an intrinsic receptor **dimerization** site and suggest that similar sites may exist in other receptors.
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't
Antibodies, Monoclonal: IM, immunology
Base Sequence
Binding Sites
Cells, Cultured
Enzyme Activation
Epitope Mapping
*Hematopoietic Cell Growth Factors: ME, metabolism
Ligands
Mice
Models, Molecular
Molecular Sequence Data
Mutation
Proto-Oncogene Proteins: CH, chemistry
Proto-Oncogene Proteins: IM, immunology
*Proto-Oncogene Proteins: ME, metabolism
Receptor Protein-Tyrosine Kinases: CH, chemistry
Receptor Protein-Tyrosine Kinases: IM, immunology
***Receptor Protein-Tyrosine Kinases: ME, metabolism**
Receptors, Colony-Stimulating Factor: CH, chemistry
Receptors, Colony-Stimulating Factor: IM, immunology
*Receptors, Colony-Stimulating Factor: ME, metabolism
***Signal Transduction**
Solubility
CN EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Antibodies, Monoclonal); 0 (Hematopoietic Cell Growth Factors); 0 (Ligands); 0 (Proto-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); **0 (Stem Cell Factor)**

L113 ANSWER 37 OF 47 MEDLINE
AN 95081116 MEDLINE

DN 95081116
TI Convergence of signaling by interleukin-3, granulocyte-macrophage colony-stimulating factor, and mast cell growth factor on JAK2 tyrosine kinase.
AU Brizzi M F; Zini M G; Aronica M G; Blechman J M; Yarden Y; Pegoraro L
CS Dipartimento di Scienze Biomediche e Oncologia Umana, Universit`a di Torino, Italy.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Dec 16) 269 (50) 31680-4. Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199503
AB Mast cell growth factor (MGF) (also called **stem cell factor**) synergizes with several lymphokines, including interleukin-3 (IL-3) and granulocyte-macrophage colony-stimulating factor (GM-CSF), to promote proliferation and differentiation of certain hemopoietic progenitor cells. Although similar patterns of tyrosine-phosphorylated proteins characterize cells stimulated by MGF, IL-3, and GM-CSF, only the MGF receptor is a tyrosine kinase, and the **heterodimeric** receptors for IL-3 and GM-CSF share a common beta subunit that is devoid of enzymatic activity. Here we show that signaling pathways utilized by all three cytokines include the cytoplasmic tyrosine kinase JAK2. Analysis of several factor-dependent myeloid cell lines indicated that JAK2 is physically associated with the common beta subunit and with MGF receptor (c-**Kit**) even prior to **ligand binding**. However, each of the **ligands** induced elevated tyrosine phosphorylation of JAK2 and a consequent increase in its catalytic activity. These results demonstrate for the first time the convergence within the same myeloid cells of signaling pathways originating in two distinct lymphokine receptors and a tyrosine kinase receptor on activation of a cytoplasmic tyrosine kinase.
CT Check Tags: Human; In Vitro; Support, Non-U.S. Gov't
Amino Acid Sequence
*Granulocyte-Macrophage Colony-Stimulating Factor: PH, physiology
*Hematopoietic Cell Growth Factors: PH, physiology
*Interleukin-3: PH, physiology
Molecular Sequence Data
Peptides: CH, chemistry
Peptides: IM, immunology
***Protein-Tyrosine Kinase: ME, metabolism**
*Proto-Oncogene Proteins: ME, metabolism
***Receptor Protein-Tyrosine Kinases: ME, metabolism**
*Receptors, Colony-Stimulating Factor: ME, metabolism
Signal Transduction
Tyrosine: AA, analogs & derivatives
Tyrosine: ME, metabolism
RN 21820-51-9 (Phosphotyrosine); 55520-40-6 (Tyrosine); 83869-56-1 (Granulocyte-Macrophage Colony-Stimulating Factor)
CN EC 2.7.1.- (Janus kinase 2); EC 2.7.1.112 (Protein-Tyrosine Kinase); EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Hematopoietic Cell Growth Factors); 0 (Interleukin-3); 0 (Peptides); 0 (Proto-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); **0 (Stem Cell Factor)**
GEN c-kit

L113 ANSWER 38 OF 47 MEDLINE
AN 95014303 MEDLINE
DN 95014303
TI The ubiquitously expressed Syp phosphatase interacts with c-kit and Grb2 in hematopoietic cells.
AU Tauchi T; Feng G S; Marshall M S; Shen R; Mantel C; Pawson T; Broxmeyer H E
CS Department of Medicine (Hematology/Oncology), Indiana University School of

Medicine, Indianapolis 46202.
NC R37 CA36464 (NCI)
R01 HL46549 (NHLBI)
R01 HL49202 (NHLBI)
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Oct 7) 269 (40) 25206-11.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199501
AB The c-kit proto-oncogene encodes a transmembrane tyrosine kinase receptor, which is important for the normal development of hematopoietic cells, melanoblasts, and germ cells. Autophosphorylation of c-kit receptor on tyrosine creates **binding** sites for cellular src homology 2 (SH2)-containing signaling molecules. The discovery of phosphotyrosine phosphatases that contain SH2 domains suggests roles for these molecules in growth factor signaling pathways. We found that Syp, a phosphotyrosine phosphatase widely expressed in all the tissues in mammals, associates with c-kit receptor after activation with its **ligand**, steel factor, in the factor-dependent cell line, M07e. Both NH2-terminal and COOH-terminal SH2 domains of Syp, made as glutathione S-transferase fusion proteins, were able to **bind** to the activated c-kit receptor in vitro. Furthermore, Syp became marginally phosphorylated on tyrosine upon c-kit receptor activation, and tyrosine-phosphorylated Syp was found to be complexed with Grb2 in steel factor-stimulated M07e cells. Direct **binding** between Syp and Grb2 was also observed in vitro. Last, Ras and Raf interacts in vitro as a result of steel factor-stimulated Ras activation. These results suggest that Syp may be an important signaling component downstream of the c-kit receptor and involved in activation of the Ras signaling pathway in hematopoietic cells.
CT Check Tags: Human; Support, U.S. Gov't, P.H.S.
Cell Line
Hematopoietic Cell Growth Factors: PD, pharmacology
Phosphorylation
Protein-Serine-Threonine Kinases: ME, metabolism
***Protein-Tyrosine-Phosphatase: ME, metabolism**
*Proteins: ME, metabolism
Proto-Oncogene Protein p21(ras): ME, metabolism
*Proto-Oncogene Proteins: ME, metabolism
***Receptor Protein-Tyrosine Kinases: ME, metabolism**
*Receptors, Colony-Stimulating Factor: ME, metabolism
Signal Transduction
CN EC 2.7.10 (Protein-Serine-Threonine Kinases); EC 2.7.10.- (Proto-Oncogene Proteins c-raf); EC 2.7.11.- (Proto-Oncogene Protein c-kit); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); EC 3.1.3.- (Syp protein); EC 3.1.3.48 (Protein-Tyrosine-Phosphatase); EC 3.6.1.- (Proto-Oncogene Protein p21(ras)); 0 (growth factor receptor-bound protein-2); 0 (Hematopoietic Cell Growth Factors); 0 (Proteins); 0 (Proto-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); **0 (Stem Cell Factor)**

L113 ANSWER 39 OF 47 MEDLINE
AN 94325604 MEDLINE
DN 94325604
TI The kit-ligand (steel factor) and its receptor c-kit/W: pleiotropic roles in gametogenesis and melanogenesis.
AU Besmer P; Manova K; Duttlinger R; Huang E J; Packer A; Gyssler C; Bachvarova R F
CS Molecular Biology Program Sloan-Kettering Institute, New York, NY.
SO DEVELOPMENT. SUPPLEMENT, (1993) 125-37. Ref: 91
Journal code: A8Z. ISSN: 0950-1991.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English

FS Priority Journals
EM 199411
AB The c-kit receptor tyrosine kinase belongs to the PDGF/CSF-1/c-kit receptor subfamily. The kit-ligand, KL, also called steel factor, is synthesized from two alternatively spliced mRNAs as transmembrane proteins that can either be proteolytically cleaved to produce soluble forms of KL or can function as cell-associated molecules. The c-kit receptor kinase and KL are encoded at the white spotting (W) and steel (Sl) loci of the mouse, respectively. Mutations at both the W and the Sl locus cause deficiencies in gametogenesis, melanogenesis and hematopoiesis. The c-kit receptor is expressed in the cellular targets of W and Sl mutations, while KL is expressed in their microenvironment. In melanogenesis, c-kit is expressed in melanoblasts from the time they leave the neural crest and expression continues during embryonic development and in the melanocytes of postnatal animals. In gametogenesis c-kit is expressed in primordial germ cells, in spermatogonia, and in primordial and growing oocytes, implying a role at three distinct stages of gametogenesis. Many mutant alleles are known at W and Sl loci and their phenotypes vary in the degree of severity in the different cellular targets of the mutations. While many W and Sl alleles severely affect primordial germ cells (PGC), several mild Sl alleles have weak effects on PGCs and exhibit differential male or female sterility. Steel Panda (Sl(pan)) is a KL expression mutation in which KL RNA transcript levels are reduced in most tissues analyzed. In female Sl(pan)/Sl(pan) mice, ovarian follicle development is arrested at the one layered cuboidal stage as a result of reduced KL expression in follicle cells, indicating a role for c-kit in oocyte growth. Wsh is a c-kit expression mutation, which affects mast cells and melanogenesis. While the mast cell defect results from lack of c-kit expression, the pigmentation deficiency appears to stem from ectopic c-kit receptor expression in the somitic dermatome at the time of migration of melanoblasts from the neural crest to the periphery. It is proposed that the ectopic c-kit expression in Wsh mice affects early melanogenesis in a dominant fashion. The "sash" or white belt of Wsh/+ animals and some other mutant mice is explained by the varying density of melanoblasts along the body axis of wild-type embryos.
CT Check Tags: Animal; Female; Human; Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
***Gametogenesis: GE, genetics**
*Hematopoietic Cell Growth Factors: GE, genetics
Mice
Mutation: PH, physiology
Phenotype
***Pigmentation: GE, genetics**
*Proto-Oncogene Proteins: GE, genetics
***Receptor Protein-Tyrosine Kinases: GE, genetics**
*Receptors, Colony-Stimulating Factor: GE, genetics
***Signal Transduction: GE, genetics**
CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Hematopoietic Cell Growth Factors); 0 (Proto-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); **0 (Stem Cell Factor)**

L113 ANSWER 40 OF 47 MEDLINE
AN 94239532 MEDLINE
DN 94239532
TI Cytoplasmic domains of the interleukin-2 receptor beta and gamma chains mediate the signal for T-cell proliferation.
AU Nelson B H; Lord J D; Greenberg P D
CS Fred Hutchinson Cancer Research Center, Seattle, Washington 98104.
SO NATURE, (1994 May 26) 369 (6478) 333-6.
Journal code: NSC. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199408

AB The interleukin-2 receptor (IL-2R) consists of three distinct chains (alpha, beta, gamma) which bind IL-2 and generate a proliferative signal in T cells. To define the mechanism of receptor activation, chimaeric receptors were constructed from the intracellular region of either IL-2R beta or IL-2R gamma and the extracellular region of c-kit, a receptor tyrosine kinase that homodimerizes on binding stem cell factor (SCF). We report here that binding of SCF to the beta-chain chimaera induced proliferation of the pro-B-cell line BA/F3, but not T cells. But in T cells expressing both the beta- and gamma-chain chimaeras, SCF induced proliferation and tyrosine phosphorylation characteristic of the native IL-2R signal. Chimaeric IL-2 receptor beta and gamma chains constructed with the heterodimeric extracellular regions of the granulocyte-macrophage colony stimulating factor receptor (GM-CSFR) also provided the IL-2R signal. Thus, heterodimerization of the cytoplasmic domains of IL-2R beta and -gamma appears necessary and sufficient for signalling in T cells.

CT Check Tags: Animal; Human
B-Lymphocytes: PH, physiology
Base Sequence
Biopolymers
Cell Line
Chimeric Proteins
Granulocyte-Macrophage Colony-Stimulating Factor: ME, metabolism
Hematopoietic Cell Growth Factors: ME, metabolism
Lymphocyte Transformation: IM, immunology
Mice
Molecular Sequence Data
Phosphorylation
Proto-Oncogene Proteins: PH, physiology
Receptor Protein-Tyrosine Kinases: PH, physiology
Receptors, Colony-Stimulating Factor: PH, physiology
*Receptors, Interleukin-2: CH, chemistry
*Receptors, Interleukin-2: PH, physiology
***Signal Transduction: IM, immunology**
*T-Lymphocytes: PH, physiology
Tyrosine: ME, metabolism

RN 55520-40-6 (Tyrosine); 83869-56-1 (Granulocyte-Macrophage Colony-Stimulating Factor)

CN EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Biopolymers); 0 (Chimeric Proteins); 0 (Hematopoietic Cell Growth Factors); 0 (Proto-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); 0 (Receptors, Interleukin-2); **0 (Stem Cell Factor)**

L113 ANSWER 41 OF 47 MEDLINE
AN 94171899 MEDLINE
DN 94171899
TI Epitope mapping and functional studies with three monoclonal antibodies to the c-kit receptor tyrosine kinase, YB5.B8, 17F11, and SR-1.
AU Ashman L K; Buhring H J; Aylett G W; Broudy V C; Muller C
CS Leukaemia Research Unit, Hanson Centre for Cancer Research, Adelaide, South Australia.
NC DK 44194 (NIDDK)
SO JOURNAL OF CELLULAR PHYSIOLOGY, (1994 Mar) 158 (3) 545-54.
Journal code: HNB. ISSN: 0021-9541.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199406
AB Three monoclonal antibodies (MAbs) to the human c-kit receptor tyrosine kinase (P145c-kit), derived in independent laboratories, have been extensively used in studies of c-kit expression and the role of its ligand, steel factor (SLF), in hemopoiesis and mast cell differentiation and function. In this study, the relationship between the epitopes they identify, and their effects on SLF binding,

receptor internalization, and signal transduction are compared. Epitope mapping studies carried out on the high P145c-kit-expressing cell line HEL-DR showed that SR-1 identifies an epitope independent of those bound by YB5.B8 and 17F11, while the latter two antibodies bound to distinct but interacting epitopes. SR-1 potently blocked the binding of SLF to P145c-kit on these cells and also on cells of the factor-dependent line MO7e. In contrast, YB5.B8 and 17F11 had minimal effects on ligand binding. Conversely, SLF partially blocked the binding of SR-1 and YB5.B8 to cells, while binding of 17F11 was actually enhanced by SLf on some target cells. Preincubation of HEL-DR and MO7e cells with MAbs prior to exposure to SLF revealed that 17F11 itself brought about partial down-regulation of P145c-kit and did not inhibit SLF-mediated down-regulation. SR-1 caused minimal down-regulation and inhibited SLF-mediated receptor internalization. YB5.B8 had minimal effects on either cell line in this assay. To determine whether the antibodies had any agonist activity, they were compared with SLF for their ability to bring about receptor phosphorylation in intact MO7e cells. All three antibodies induced detectable tyrosine phosphorylation with 17F11 being the most effective, while YB5.B8 was the least effective. Finally, the ability of the antibodies to influence the proliferation of the MO7e cells was examined. As expected, SR-1 potently inhibited the proliferative response to SLF, while 17F11 weakly inhibited and YB5.B8 had negligible effect. In the absence of SLF both 17F11 and YB5.B8 displayed very weak but reproducible agonist activity.

CT Check Tags: Animal; Comparative Study; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
*Antibodies, Monoclonal: IM, immunology
Antibodies, Monoclonal: ME, metabolism
Antibodies, Monoclonal: PD, pharmacology
Binding Sites, Antibody
Binding, Competitive
Bone Marrow: CY, cytology
Bone Marrow: ME, metabolism
Bone Marrow: UL, ultrastructure
Cells, Cultured
CHO Cells
Endocytosis
*Epitopes: IM, immunology
Flow Cytometry
Fluorescent Antibody Technique
Hamsters
Hematopoietic Cell Growth Factors: ME, metabolism
Leukemia, Erythroblastic, Acute: ME, metabolism
Leukemia, Erythroblastic, Acute: PA, pathology
Leukemia, Megakaryocytic, Acute: ME, metabolism
Leukemia, Megakaryocytic, Acute: PA, pathology
*Peptide Mapping
Phosphorylation
*Proto-Oncogene Proteins: IM, immunology
Proto-Oncogene Proteins: ME, metabolism
*Proto-Oncogene Proteins: PH, physiology
***Receptor Protein-Tyrosine Kinases: IM, immunology**
Receptor Protein-Tyrosine Kinases: ME, metabolism
***Receptor Protein-Tyrosine Kinases: PH, physiology**
*Receptors, Colony-Stimulating Factor: IM, immunology
Receptors, Colony-Stimulating Factor: ME, metabolism
*Receptors, Colony-Stimulating Factor: PH, physiology
Signal Transduction: PH, physiology
Tumor Cells, Cultured

CN EC 2.7.11.- (Proto-Oncogene Protein c-kit); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Antibodies, Monoclonal); 0 (Binding Sites, Antibody); 0 (Epitopes); 0 (Hematopoietic Cell Growth Factors); 0 (Proto-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); **0 (Stem Cell Factor)**

L113 ANSWER 42 OF 47 MEDLINE

AN 94105123 MEDLINE
DN 94105123
TI Ligand-induced activation of chimeric receptors between the erythropoietin receptor and receptor tyrosine kinases.
AU Ohashi H; Maruyama K; Liu Y C; Yoshimura A
CS Pharmaceutical Laboratories, Kirin Brewery Co. LTD., Gunma, Japan.
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1994 Jan 4) 91 (1) 158-62.
Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199404
AB Ligand-induced **dimerization** is a key step in the activation of receptor tyrosine kinases, including the epidermal growth factor receptor, **stem cell factor** receptor (c-**kit**), and colony-stimulating factor 1 receptor (c-**fms**). The erythropoietin receptor (EPOR), a member of the cytokine receptor family, contains no kinase motif and its activation mechanism remains unclear. Here we show that chimeric receptors carrying the extracellular domain of the epidermal growth factor receptor or c-**kit** linked to the cytoplasmic domain of the EPOR, transmitted epidermal growth factor or **stem cell factor**-dependent proliferation signals in an interleukin 3-dependent cell line. The chimeric receptors as well as the wild-type EPOR also mediated the ligand-induced tyrosine phosphorylation of a set of similar proteins. Moreover, erythropoietin triggered mitogenic signals of chimeric receptors carrying the extracellular domain of the EPOR linked to the tyrosine kinase of c-fms. These data demonstrate the interchangeability of domains between two distinct receptor families and suggest that ligand-induced **dimerization** is a key step in activating the EPOR.
CT Check Tags: Animal; Support, Non-U.S. Gov't
Amino Acid Sequence
Base Sequence
Cell Line
Chimeric Proteins: ME, metabolism
Enzyme Activation
Mice
Mitosis
Molecular Sequence Data
Oligodeoxyribonucleotides: CH, chemistry
Proto-Oncogene Proteins: CH, chemistry
***Receptor Protein-Tyrosine Kinases: CH, chemistry**
Receptor, Epidermal Growth Factor: CH, chemistry
Receptor, Macrophage Colony-Stimulating Factor: CH, chemistry
Receptors, Colony-Stimulating Factor: CH, chemistry
*Receptors, Erythropoietin: CH, chemistry
Signal Transduction
Structure-Activity Relationship
Transfection
Tyrosine: AA, analogs & derivatives
Tyrosine: ME, metabolism
RN 21820-51-9 (Phosphotyrosine); 55520-40-6 (Tyrosine)
CN EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); EC 2.7.11.- (Receptor, Epidermal Growth Factor); EC 2.7.11.- (Receptor, Macrophage Colony-Stimulating Factor); 0 (Chimeric Proteins); 0 (Oligodeoxyribonucleotides); 0 (Proto-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); 0 (Receptors, Erythropoietin)
GEN EGFR; EPOR; c-kit; c-fms

L113 ANSWER 43 OF 47 MEDLINE
AN 94004531 MEDLINE
DN 94004531
TI Molecular genetic approaches to the elucidation of hematopoietic stem cell

function.
AU Bernstein A
CS Samuel Lunenfeld Research Institute, Mount Sinai Hospital, Toronto, Ontario, Canada.
SO STEM CELLS, (1993 Jul) 11 Suppl 2 31-5. Ref: 33
Journal code: BN2. ISSN: 1066-5099.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199401
AB The past few years have seen considerable advances in the development of the methodologies for discovering novel genes critical to hematopoietic stem cell function and for analyzing their biological role in hematopoiesis. This review briefly discusses some common themes that are emerging from the molecular genetic approaches to hematopoietic stem cell function.
CT Check Tags: Animal; Support, Non-U.S. Gov't
Anemia: GE, genetics
Germ Cells: DE, drug effects
*Hematopoiesis: GE, genetics
Hematopoietic Cell Growth Factors: GE, genetics
Hematopoietic Cell Growth Factors: PD, pharmacology
*Hematopoietic Cell Growth Factors: PH, physiology
Hematopoietic Stem Cells: DE, drug effects
*Hematopoietic Stem Cells: PH, physiology
Infertility: GE, genetics
Melanocytes: DE, drug effects
Mice
Mice, Mutant Strains: GE, genetics
Pigmentation Disorders: GE, genetics
Protein-Tyrosine Kinase: PH, physiology
Proto-Oncogene Proteins: GE, genetics
*Proto-Oncogene Proteins: PH, physiology
Receptor Protein-Tyrosine Kinases: GE, genetics
***Receptor Protein-Tyrosine Kinases: PH, physiology**
Receptors, Cell Surface: PH, physiology
Receptors, Colony-Stimulating Factor: GE, genetics
*Receptors, Colony-Stimulating Factor: PH, physiology
Signal Transduction
CN EC 2.7.1.112 (Protein-Tyrosine Kinase); EC 2.7.11.- (Proto-Oncogene Protein c-kit); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Hematopoietic Cell Growth Factors); 0 (Proto-Oncogene Proteins); 0 (Receptors, Cell Surface); 0 (Receptors, Colony-Stimulating Factor); **0 (Stem Cell Factor)**
GEN Sl; c-myb; c-src; c-fyn; c-kit; src; GATA-1

L113 ANSWER 44 OF 47 MEDLINE
AN 94004519 MEDLINE
DN 94004519
TI Structure-function analyses of the kit receptor for the steel factor.
AU Blechman J M; Lev S; Givol D; Yarden Y
CS Department of Chemical Immunology, Weizmann Institute of Science, Rehovot, Israel.
SO STEM CELLS, (1993 Jul) 11 Suppl 2 12-21. Ref: 42
Journal code: BN2. ISSN: 1066-5099.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199401

AB **Binding** of the Steel factor (SLF) to the product of the c-**kit** proto-oncogene stimulates the receptor's intrinsic tyrosine kinase that phosphorylates a set of cytoplasmic signaling molecules. Germ-line mutations in the genes that encode the receptor or the **ligand** result in remarkably similar phenotypes that affect melanogenesis, erythropoiesis and gametogenesis in mice. We concentrated on the initial events of the signal transduction pathway that underlies these processes. The extracellular portion **of Kit** is comprised of five immunoglobulin-(Ig)-like domains. **Ligand binding** to this domain induces rapid and extensive **dimerization** of the receptor molecules in a mechanism that involves monovalent **binding** of the **dimeric ligand**, followed by an increase in receptors' affinity and gradual stabilization of the **dimers**. It thus appears that **Kit** has at least two functions: **ligand binding** and **ligand**-induced receptor **dimerization**, in addition to the kinase activity. Both functions are independent of the transmembrane and cytoplasmic domains, **as** a recombinant soluble ectodomain retained high affinity to SLF and **ligand**-dependent **dimerization**. In order to correlate these functions with specific structures, we employed **ligand**-competitive monoclonal antibodies, soluble deletion mutants of the ectodomain and chimeric human-mouse **Kit** proteins. These approaches indicated that the N-terminal three Ig-like domains constitute the **binding** site, whose core is the second domain. Further experiments suggested that a putative **dimerization** site is distinct from the **binding** cleft and may be located on the fourth Ig-like domain.

CT Check Tags: Animal; Human; Support, Non-U.S. Gov't
Antibodies, Monoclonal: IM, immunology
Binding Sites
Hematopoietic Cell Growth Factors: GE, genetics
*Hematopoietic Cell Growth Factors: ME, metabolism
Mice
Models, Molecular
Polymers
Protein Binding
Protein Conformation
Protein Engineering
Protein-Tyrosine Kinase: ME, metabolism
Proto-Oncogene Proteins: CH, chemistry
Proto-Oncogene Proteins: GE, genetics
Proto-Oncogene Proteins: IM, immunology
*Proto-Oncogene Proteins: ME, metabolism
Proto-Oncogenes
Receptor Protein-Tyrosine Kinases: CH, chemistry
Receptor Protein-Tyrosine Kinases: GE, genetics
Receptor Protein-Tyrosine Kinases: IM, immunology
***Receptor Protein-Tyrosine Kinases: ME, metabolism**
Receptors, Colony-Stimulating Factor: CH, chemistry
Receptors, Colony-Stimulating Factor: GE, genetics
Receptors, Colony-Stimulating Factor: IM, immunology
*Receptors, Colony-Stimulating Factor: ME, metabolism
Recombinant Fusion Proteins: ME, metabolism
Signal Transduction
Structure-Activity Relationship

CN EC 2.7.1.112 (Protein-Tyrosine Kinase); EC 2.7.11.- (**Proto-Oncogene Protein c-kit**); EC 2.7.11.- (Receptor Protein-Tyrosine Kinases); 0 (Antibodies, Monoclonal); 0 (Hematopoietic Cell Growth Factors); 0 (Polymers); 0 (Proto-Oncogene Proteins); 0 (Receptors, Colony-Stimulating Factor); 0 (Recombinant Fusion Proteins); **0 (Stem Cell Factor)**

GEN c-kit; Sl; v-kit

L113 ANSWER 45 OF 47 MEDLINE
AN 92313795 MEDLINE
DN 92313795
TI The kit receptor and its ligand, steel factor, as regulators of

hemopoiesis.

AU Broxmeyer H E; Maze R; Miyazawa K; Carow C; Hendrie P C; Cooper S; Hangoc G; Vadhan-Raj S; Lu L

CS Department of Medicine (Hematology/Oncology), Indiana University School of Medicine, Indianapolis 46202.

NC R37 CA36464 (NCI)
R01 HL46549 (NHLBI)
R01 CA36740 (NCI)
+

SO CANCER CELLS, (1991 Dec) 3 (12) 480-7. Ref: 68
Journal code: AU5. ISSN: 1042-2196.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 199210

AB Mouse strains carrying mutations at the Dominant White Spotting (W) locus or the Steel (Sl) locus are anemic and display defects in pigmentation and gametogenesis. In W mutants the anemia is due to a deficiency of hemopoietic stem cells and, in Sl mutants, to a deficiency of supporting stromal cells in the bone marrow. The W locus encodes the c-kit proto-oncogene product, a cell surface receptor with protein-tyrosine kinase activity, and the Sl locus encodes its ligand, a hemopoietic cytokine known variously as Steel factor (SLF), mast cell growth factor, **stem cell factor**, and Kit ligand. SLF can synergize with a number of other cytokines to stimulate growth of hemopoietic progenitors in vitro and stimulates blood cell production in vivo in animals. Here we review the biological activities of SLF, with particular emphasis on its effects on hemopoietic stem and progenitor cells. We also discuss present knowledge of the molecules involved in SLF-triggered signal transduction, and speculate on potential therapeutic applications for SLF in human disease.

CT Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Anemia: DT, drug therapy
Anemia: GE, genetics
Bone Marrow: EM, embryology
Cell Differentiation
Cell Movement
Drug Screening
Gene Expression Regulation
*Hematopoiesis: PH, physiology
Hematopoietic Cell Growth Factors: GE, genetics
Hematopoietic Cell Growth Factors: IP, isolation & purification
Hematopoietic Cell Growth Factors: PD, pharmacology
*Hematopoietic Cell Growth Factors: PH, physiology
Hematopoietic Cell Growth Factors: TU, therapeutic use
Hematopoietic Stem Cells: DE, drug effects
Leukemia: PA, pathology
Melanocytes: CY, cytology
Mice
Mice, Mutant Strains: EM, embryology
Mice, Mutant Strains: GE, genetics
Mice, Mutant Strains: PH, physiology
Protein-Tyrosine Kinase: GE, genetics
Protein-Tyrosine Kinase: ME, metabolism
Proto-Oncogene Proteins: GE, genetics
*Proto-Oncogene Proteins: PH, physiology
Proto-Oncogenes
Rats
Signal Transduction
Tumor Stem Cells: DE, drug effects
Tumor Stem Cells: PA, pathology

CN EC 2.7.1.112 (Protein-Tyrosine Kinase); EC 2.7.11.- (Proto-Oncogene

Protein c-kit); 0 (Hematopoietic Cell Growth Factors); 0 (Proto-Oncogene Proteins); **0 (Stem Cell Factor)**
GEN c-kit; W; Sl; c-fms; Sld; C-KIT; KIT; SLF

L113 ANSWER 46 OF 47 MEDLINE
AN 91246171 MEDLINE
DN 91246171
TI The Steel/W transduction pathway: kit autophosphorylation and its association with a unique subset of cytoplasmic signaling proteins is induced by the Steel factor.
AU Rottapel R; Reedijk M; Williams D E; Lyman S D; Anderson D M; Pawson T; Bernstein A
CS Division of Molecular and Developmental Biology, Samuel Lunenfeld Research Institute of Mount Sinai Hospital, Toronto, Ontario, Canada.
SO MOLECULAR AND CELLULAR BIOLOGY, (1991 Jun) 11 (6) 3043-51.
Journal code: NGY. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199109
AB The W/c-kit and Steel loci respectively encode a receptor tyrosine kinase (Kit) and its extracellular **ligand**, Steel factor, which are essential for the development of hematopoietic, melanocyte, and germ cell lineages in the mouse. To determine the biochemical basis of the Steel/W developmental pathway, we have investigated the response of the Kit tyrosine kinase and several potential cytoplasmic targets to stimulation with Steel in mast cells derived from normal and mutant W mice. In normal mast cells, Steel induces Kit to autophosphorylate on tyrosine and **bind** to phosphatidylinositol 3'-kinase (PI3K) and phospholipase C-gamma 1 but not detectably to Ras GTPase-activating protein. Additionally, we present evidence that Kit tyrosine phosphorylation acts as a switch to promote complex formation with PI3K. In mast cells from mice homozygous for the W42 mutant allele, Kit is not tyrosine phosphorylated and fails to **bind** PI3K following Steel stimulation. In contrast, in the transformed mast cell line P815, Kit is constitutively phosphorylated and **binds** to PI3K in the absence of **ligand**. These results suggest that Kit autophosphorylation and its physical association with a unique subset of cytoplasmic signaling proteins are critical for mammalian development.
CT Check Tags: Animal; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Blotting, Western
Cell Line
*Hematopoietic Cell Growth Factors: GE, genetics
Hematopoietic Cell Growth Factors: ME, metabolism
*Hematopoietic Stem Cells: PH, physiology
Homozygote
Mast Cells: PH, physiology
Mice
Mice, Inbred C57BL
Mice, Mutant Strains
Mutation
Phospholipase C: ME, metabolism
Phosphorylation
Phosphotransferases: ME, metabolism
Protein Binding
***Protein-Tyrosine Kinase: GE, genetics**
Protein-Tyrosine Kinase: ME, metabolism
Proteins: ME, metabolism
*Proto-Oncogene Proteins: GE, genetics
Proto-Oncogene Proteins: ME, metabolism
*Proto-Oncogenes
Recombinant Fusion Proteins: ME, metabolism
Restriction Mapping
***Signal Transduction**
CN EC 2.7 (Phosphotransferases); EC 2.7.1.112 (Protein-Tyrosine Kinase); EC

2.7.1.137 (1-Phosphatidylinositol 3-Kinase); EC 2.7.11.- (Proto-Oncogene Protein c-kit); EC 3.1.4.3 (Phospholipase C); 0 (ras GTPase-Activating Proteins); 0 (GTPase-Activating Proteins); 0 (Hematopoietic Cell Growth Factors); 0 (Proto-Oncogene Proteins); 0 (Recombinant Fusion Proteins); **0 (Stem Cell Factor)**

L113 ANSWER 47 OF 47 MEDLINE
AN 91160520 MEDLINE
DN 91160520
TI A specific combination of substrates is involved in signal transduction by the kit-encoded receptor.
AU Lev S; Givol D; Yarden Y
CS Department of Chemical Immunology, Weizmann Institute of Science, Rehovot, Israel.
NC 1 RO1 CA512712 (NCI)
SO EMBO JOURNAL, (1991 Mar) 10 (3) 647-54.
Journal code: EMB. ISSN: 0261-4189.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199106
AB The kit protooncogene encodes a transmembrane tyrosine kinase related to the receptors for the platelet derived growth factor (PDGF-R) and the macrophage growth factor (CSF1-R), and was very recently shown to **bind** a **stem cell factor**. To compare signal transduction by the kit kinase with signaling by homologous receptors we constructed a chimeric protein composed of the extracellular domain of the epidermal growth factor receptor (EGF-R) and the transmembrane and cytoplasmic domains of kit. We have previously shown that the chimeric receptor transmits potent mitogenic and transforming signals in response to the heterologous **ligand**. Here we demonstrate that upon **ligand binding**, the **ligand**-receptor complex undergoes endocytosis and degradation and induces short- and long-term cellular effects. Examination of the signal transduction pathway revealed that the activated kit kinase strongly associates with phosphatidylinositol 3'-kinase activity and a phosphoprotein of 85 kd. In addition, the **ligand**-stimulated kit kinase is coupled to modifications of phospholipase C gamma and the Raf1 protein kinase. However, it does not lead to a significant change in the production of inositol phosphate. Comparison of our results with the known signaling pathways of PDGF-R and CSF1-R suggests that each receptor is coupled to a specific combination of signal transducers.
CT Check Tags: Animal; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Biological Transport, Active: DE, drug effects
Cell Line
Chimera
Deoxyglucose: ME, metabolism
Endocytosis
Epidermal Growth Factor: PD, pharmacology
Kinetics
Ligands
Macrophage Colony-Stimulating Factor: PD, pharmacology
Mice
Models, Biological
Platelet-Derived Growth Factor: ME, metabolism
***Protein-Tyrosine Kinase: GE, genetics**
*Proto-Oncogene Proteins: GE, genetics
Proto-Oncogene Proteins: ME, metabolism
*Proto-Oncogenes
*Receptors, Cell Surface: GE, genetics
Receptors, Cell Surface: ME, metabolism
***Signal Transduction**
RN 154-17-6 (Deoxyglucose); 62229-50-9 (Epidermal Growth Factor); 81627-83-0 (Macrophage Colony-Stimulating Factor)
CN EC 2.7.1.112 (Protein-Tyrosine Kinase); EC 2.7.11.- (Proto-Oncogene

Protein c-kit); EC 2.7.11.- (Receptors, Platelet-Derived Growth Factor); 0 (Ligands); 0 (Platelet-Derived Growth Factor); 0 (Proto-Oncogene Proteins); 0 (Receptors, Cell Surface)
GEN kit

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L118 1 S L117 AND LONGLEY ?/AU
L119 236 S L117 AND (185? OR *355? OR *1400? OR *1600? OR 1650?)/CC
L120 48 S L119 AND *34508/CC
L121 40 S L120 AND 150?/CC
L122 41 S L118,L121
L123 8 S L120 NOT L122
L124 49 S L122,L123

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L124 ANSWER 1 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1999:229797 BIOSIS
DN PREV199900229797
TI Transgenic mice expressing **stem cell factor** in basal keratinocytes develop postinflammatory hyperpigmentation in response to irritant and allergic contactants.
AU Carter, E. L. (1); Tigelaar, R. E.; **Longley, B. J.**
CS (1) Department of Dermatology, Columbia University, New York, NY USA
SO Journal of Investigative Dermatology, (**April, 1999**) Vol. 112, No. 4, pp. 539.
Meeting Info.: **60th Annual Meeting of the Society for Investigative Dermatology** Chicago, Illinois, USA May 5-9, 1999
ISSN: 0022-202X.
DT **Conference**
LA English
CC Integumentary System - General; Methods *18501
Cytology and Cytochemistry - Animal *02506
Biochemical Studies - General *10060
Blood, Blood-Forming Organs and Body Fluids - General; Methods *15001
Immunology and Immunochemistry - General; Methods *34502
Allergy *35500
Toxicology - General; Methods and Experimental *22501
Endocrine System - General *17002

General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
BC Muridae 86375
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Dermatology (Human Medicine, Medical Sciences)
IT Parts, Structures, & Systems of Organisms
basal keratinocyte: integumentary system; epidermis: integumentary system
IT Diseases
postinflammatory hyperpigmentation: integumentary system disease
IT Chemicals & Biochemicals
allergic contactant: allergen; irritant contactant: toxin; keratin 14 promoter; **stem cell factor**: expression
IT Miscellaneous Descriptors
Meeting Abstract
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae): model, transgenic
ORGN Organism Superterms
Animals; Chordates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates

L124 ANSWER 2 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1999:136245 BIOSIS
DN PREV199900136245
TI **Stem cell factor** (SCF) stimulates adhesion of human intestinal mast cells to extracellular matrix proteins.
AU Lorentz, A. (1); Sellge, G. (1); Manns, M. P. (1); Schuppan, D.; Levi-Schaffer, F.; Bischoff, S. C. (1)
CS (1) Dep. Gastroenterology and Hepatology, Med. Sch. Hannover, Hannover Germany
SO Journal of Allergy and Clinical Immunology, (**Jan., 1999**) Vol. 103, No. 1 PART 2, pp. S41.
Meeting Info.: **55th Annual Meeting of the American Academy of Allergy, Asthma and Immunology** Orlando, Florida, USA February 26-March 3, 1999 American Academy of Allergy, Asthma, and Immunology . ISSN: 0091-6749.
DT **Conference**
LA English
CC **Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508**
Cytology and Cytochemistry - Human *02508
Digestive System - Physiology and Biochemistry *14004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
BC Hominidae 86215
IT Major Concepts
Immune System (Chemical Coordination and Homeostasis)
IT Parts, Structures, & Systems of Organisms
intestinal mast cells: adhesion, blood and lymphatics, digestive system, immune system
IT Chemicals & Biochemicals
extracellular matrix proteins; fibronectin: matric protein; **stem cell factor**
IT Miscellaneous Descriptors
Meeting Abstract; Meeting Poster
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms

Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L124 ANSWER 3 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1999:125314 BIOSIS
DN PREV199900125314
TI Mast cell regulation by cytokines and nitric oxide.
AU Coleman, J. W. (1)
CS (1) Dep. Pharmacol., Univ. Liverpool, Liverpool L69 3GE UK
SO Immunology, (**Dec., 1998**) Vol. 95, No. SUPPL. 1, pp. 19.
Meeting Info.: **6th Annual Congress of the British Society for Immunology** Harrogate, England, UK December 1-4, 1998
ISSN: 0019-2805.
DT **Conference**
LA English
CC **Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508**
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Endocrine System - General *17002
Allergy *35500
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
IT Major Concepts
Immune System (Chemical Coordination and Homeostasis)
IT Parts, Structures, & Systems of Organisms
mast cells: immune system
IT Chemicals & Biochemicals
cytokines; nitric oxide; **stem cell factor** ; IFN-gamma [interferon-gamma]; IL-4 [interleukin-4]
IT Miscellaneous Descriptors
allergic reactions; immunity; mast cell regulation; **Meeting Abstract**
RN 10102-43-9 (NITRIC OXIDE)

L124 ANSWER 4 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1999:125313 BIOSIS
DN PREV199900125313
TI Regulation of the mucosal mast cells response following parasitic infection.
AU Grencis, R. K. (1)
CS (1) Sch. Biol. Sci., Stopford Build., Univ. Manchester, Oxford Road, Manchester M13 9PT UK
SO Immunology, (**Dec., 1998**) Vol. 95, No. SUPPL. 1, pp. 19.
Meeting Info.: **6th Annual Congress of the British Society for Immunology** Harrogate, England, UK December 1-4, 1998
ISSN: 0019-2805.
DT **Conference**
LA English
CC Immunology, Parasitological *35000
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Digestive System - Pathology *14006
Endocrine System - General *17002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Parasitology - General *60502
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
BC Nematoda 51300
IT Major Concepts
Immune System (Chemical Coordination and Homeostasis); Parasitology
IT Parts, Structures, & Systems of Organisms
mast cells: immune system
IT Diseases
parasitic infection: parasitic disease
IT Chemicals & Biochemicals
interleukin-9 [IL-9]; intestinal mastocytosis: immune system disease; **stem cell factor**

IT Alternate Indexing
Parasitic Diseases (MeSH)
IT Miscellaneous Descriptors
immunity; mucosal mast cell response: regulation; **Meeting Abstract**
ORGN Super Taxa
Nematoda: Aschelminthes, Helminthes, Invertebrata, Animalia
ORGN Organism Name
nematode (Nematoda): intestinal parasite
ORGN Organism Superterms
Animals; Aschelminths; Helminths; Invertebrates

L124 ANSWER 5 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:248327 BIOSIS
DN PREV199800248327
TI Rapid reduction in the size of mouse cutaneous mast cell populations by apoptosis after cessation of treatment with SCF does not result in skin inflammation.
AU Maurer, Marcus (1); Galli, Stephen J.
CS (1) Dep. Pathol., Beth Israel Deaconess Med. Cent., Boston, MA USA
SO Journal of Investigative Dermatology, (**April, 1998**) Vol. 110, No. 4, pp. 634.
Meeting Info.: **Annual Meeting of the International Investigative Dermatology** Cologne, Germany May 7-10, 1998 The Society for Investigative Dermatology, Inc.
. ISSN: 0022-202X.
DT **Conference**
LA English
CC **Integumentary System - Pathology *18506**
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
BC Muridae 86375
IT Major Concepts
Immune System (Chemical Coordination and Homeostasis); Integumentary System (Chemical Coordination and Homeostasis)
IT Parts, Structures, & Systems of Organisms
mast cell: immune system; skin: integumentary system
IT Chemicals & Biochemicals
stem cell factor
IT Miscellaneous Descriptors
apoptosis; inflammation; **Meeting Abstract; Meeting Poster**
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae)
ORGN Organism Superterms
Animals; Chordates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates

L124 ANSWER 6 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:247926 BIOSIS
DN PREV199800247926
TI Human **stem cells factor** does not affect the morphology and expression of functionally relevant molecules of Langerhans cells in vitro.
AU Prignano, Francesca; Gerlini, Gianni; Pimpinelli, Nicola; Romagnoli, Paolo; Giannotti, Benvenuto
CS Dep. Anatomy Histology, Inst. Dermatol., Univ. Florence, Florence Italy
SO Journal of Investigative Dermatology, (**April, 1998**) Vol. 110, No. 4, pp. 567.
Meeting Info.: **Annual Meeting of the International Investigative**

Dermatology Cologne, Germany May 7-10, 1998 The Society for Investigative Dermatology, Inc.
. ISSN: 0022-202X.
DT **Conference**
LA English
CC **Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508**
Cytology and Cytochemistry - Human *02508
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Integumentary System - Physiology and Biochemistry *18504
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
BC Hominidae 86215
IT Major Concepts
Immune System (Chemical Coordination and Homeostasis); Integumentary System (Chemical Coordination and Homeostasis)
IT Parts, Structures, & Systems of Organisms
epidermis: integumentary system; Langerhans cells: immune system, in vitro, metabolism, morphology
IT Chemicals & Biochemicals
human **stem cell factor**
IT Miscellaneous Descriptors
Meeting Abstract; Meeting Poster
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L124 ANSWER 7 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:154607 BIOSIS
DN PREV199800154607
TI Adherence of human lung mast cells to bronchial epithelium.
AU Sanmugalingam, D.; Wardlaw, A. J.; Bradding, P.
CS Univ. Leicester, Glenfield Hosp., Leicester UK
SO Journal of Allergy and Clinical Immunology, (**Jan., 1998**) Vol. 101, No. 1 PART 2, pp. S216.
Meeting Info.: **54th Annual Meeting of the American Academy of Allergy, Asthma and Immunology** Washington, DC, USA March 13-18, 1998
American Academy of Allergy, Asthma, and Immunology
. ISSN: 0091-6749.
DT **Conference**
LA English
CC **Respiratory System - Pathology *16006**
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
BC Hominidae 86215
IT Major Concepts
Immune System (Chemical Coordination and Homeostasis); Respiratory System (Respiration)
IT Parts, Structures, & Systems of Organisms
bronchial epithelium: respiratory system; lung mast cells
IT Diseases
asthma: immune system disease, respiratory system disease

IT Chemicals & Biochemicals
alpha-4-beta-1; CD18; E-cadherin; SCF [**stem cell factor**]
IT Miscellaneous Descriptors
Meeting Abstract
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae); BEAS-2B (Hominidae): **bronchial** epithelial cell
ORGN Organism Superterms
Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L124 ANSWER 8 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:145813 BIOSIS
DN PREV199800145813
TI Cytokines and asthma.
AU Palma Carlos, A. G.; Palma Carlos, M. L.; **Conceicao**, Santos M.; Alcinda, Melo
CS Med. I Univ. Clinic, Immunol. Inst., Lisbon **Portugal**
SO Journal of Investigational Allergology & Clinical Immunology, (**Sept.-Oct., 1997**) Vol. 7, No. 5, pp. 270-273.
Meeting Info.: **Annual Meeting of the International Association of Asthmology, Western Europe Chapter: Interasma 97** Las Palmas de Gran Canaria, Canary Islands, Spain December 3-5, 1997 International Association of Asthmology
. ISSN: 1018-9068.
DT **Conference**
LA English
CC **Allergy *35500**
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Pathology, General and Miscellaneous - Therapy *12512
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Respiratory System - Pathology *16006
Endocrine System - General *17002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
BC Hominidae 86215
IT Major Concepts
Clinical Immunology (Human Medicine, Medical Sciences)
IT Diseases
asthma: cell recruitment, respiratory system disease, immune system disease, inflammation; atopy: immune system disease; respiratory allergy: immune system disease
IT Chemicals & Biochemicals
adhesion molecules; chemokines; cytokines: immunomodulation, production, network activation; **stem cell factor**; tumor necrosis factor alpha; IgE [**immunoglobulin** E]: synthesis
IT Methods & Equipment
immunomodulatory intervention: therapeutic method
IT Miscellaneous Descriptors
Meeting Paper
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae): patient
ORGN Organism Superterms
Animals; Chordates; Humans; Mammals; Primates; Vertebrates

L124 ANSWER 9 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:67444 BIOSIS

DN PREV199800067444
TI Oncostatin M (OSM) supports expansion of hematopoietic progenitors derived from the aorta gonad mesonephros (AGM) region of mouse embryo.
AU Mukoyama, Y. (1); Hara, T. (1); Xu, M.; Tamura, K.; Donovan, P. J.; Kim, H. (1); Kogo, H.; Tsuji, K.; Nakahata, T.; Miyajima, A. (1)
CS (1) Inst. Molecular Cellular Biosciences, Univ. Tokyo, Tokyo Japan
SO Blood, (**Nov. 15, 1997**) Vol. 90, No. 10 SUPPL. 1 PART 1, pp. 258A.
Meeting Info.: **39th Annual Meeting of the American Society of Hematology** San Diego, California, USA December 5-9, 1997 The American Society of Hematology
. ISSN: 0006-4971.
DT **Conference**
LA English
CC **Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002**
Cytology and Cytochemistry - Animal *02506
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Reproductive System - Physiology and Biochemistry *16504
Endocrine System - General *17002
Developmental Biology - Embryology - General and Descriptive *25502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
BC Muridae 86375
IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation)
IT Parts, Structures, & Systems of Organisms
aorta gonad mesonephros: embryonic structure; bone marrow: blood and lymphatics, immune system; hematopoietic progenitor cells: blood and lymphatics, expansion
IT Chemicals & Biochemicals
basic fibroblast growth factor; interleukin-6; oncostatin M: expression; **stem cell factor**
IT Miscellaneous Descriptors
hematopoiesis; **Meeting Abstract**
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae): embryo
ORGN Organism Superterms
Animals; Chordates; Mammals; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates
RN 106956-32-5 (ONCOSTATIN M)

L124 ANSWER 10 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:67249 BIOSIS
DN PREV199800067249
TI Cell cycling of mobilized peripheral blood stem cell subsets: Effect of mobilization regimen and impact on engraftment.
AU Lill, M. (1); Saks-Rosenthal, E.; Turner, S. A.; Chap, L.; Crooks, G.; Glaspy, J. A.
CS (1) Div. Hematol./Oncol., UCLA Sch. Med., Los Angeles, CA USA
SO Blood, (**Nov. 15, 1997**) Vol. 90, No. 10 SUPPL. 1 PART 1, pp. 213A.
Meeting Info.: **39th Annual Meeting of the American Society of Hematology** San Diego, California, USA December 5-9, 1997 The American Society of Hematology
. ISSN: 0006-4971.
DT **Conference**
LA English
CC **Blood, Blood-Forming Organs and Body Fluids - General; Methods**

***15001**
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Biochemical Studies - Carbohydrates *10068
Anatomy and Histology, General and Comparative - Regeneration and Transplantation *11107
Movement *12100
Pathology, General and Miscellaneous - Therapy *12512
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Reproductive System - General; Methods *16501
Reproductive System - Physiology and Biochemistry *16504
Reproductive System - Pathology *16506
Endocrine System - General *17002
Bones, Joints, Fasciae, Connective and Adipose Tissue - General; Methods *18001
Bones, Joints, Fasciae, Connective and Adipose Tissue - Physiology and Biochemistry *18004
Bones, Joints, Fasciae, Connective and Adipose Tissue - Pathology *18006
Pharmacology - Clinical Pharmacology *22005
Pharmacology - Blood and Hematopoietic Agents *22008
Pharmacology - Endocrine System *22016
Pharmacology - Immunological Processes and Allergy *22018
Pharmacology - Reproductive System; Implantation Studies *22028
Neoplasms and Neoplastic Agents - Immunology *24003
Neoplasms and Neoplastic Agents - Biochemistry *24006
Neoplasms and Neoplastic Agents - Therapeutic Agents; Therapy *24008
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
BC Hominidae 86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Methods and Techniques
IT Parts, Structures, & Systems of Organisms
blood: blood and lymphatics; peripheral blood stem cells: blood and lymphatics, subsets; CD34-positive cells: blood and lymphatics, immune system; CD38-positive cells: blood and lymphatics, immune system
IT Diseases
breast cancer: neoplastic disease, reproductive system disease/female
IT Chemicals & Biochemicals
stem cell factor; G-CSF [filgrastim, granulocyte-colony stimulating factor]
IT Methods & Equipment
bone marrow transplantation: therapeutic method, transplantation method
IT Miscellaneous Descriptors
cell cycling; cell processing; neutrophil engraftment; peripheral blood stem cell mobilization; platelet engraftment; **Meeting Abstract; Meeting Poster**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae): patient
ORGN Organism Superterms
Animals; Chordates; Humans; Mammals; Primates; Vertebrates
RN 121181-53-1 (FILGRASTIM)

L124 ANSWER 11 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1998:66747 BIOSIS
DN PREV199800066747
TI Reconstitution of humoral, cellular and natural immunity after transplantation of autologous hematopoietic progenitor cells to support high-dose chemotherapy.
AU Morgan, M. (1); Mawhinney, S.; Wang, J. K.; Shpall, E. J.; Curiel, T.
CS (1) Univ. Colorado Health Sciences Cent., Med. Serv., Biometrics Bone Marrow Transplant Unit, Denver, CO USA
SO Blood, (Nov. 15, 1997) Vol. 90, No. 10 SUPPL. 1 PART 1, pp. 100A.
Meeting Info.: 39th Annual Meeting of the American Society of Hematology San Diego, California, USA December 5-9, 1997 The American Society of Hematology
. ISSN: 0006-4971.
DT Conference
LA English
CC Neoplasms and Neoplastic Agents - Immunology *24003
Anatomy and Histology, General and Comparative - Regeneration and Transplantation *11107
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Reproductive System - Pathology *16506
Endocrine System - General *17002
Pharmacology - Clinical Pharmacology *22005
Pharmacology - Blood and Hematopoietic Agents *22008
Pharmacology - Endocrine System *22016
Pharmacology - Reproductive System; Implantation Studies *22028
Neoplasms and Neoplastic Agents - Therapeutic Agents; Therapy *24008
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals *00520
Cytology and Cytochemistry - Human *02508
Biochemical Studies - General *10060
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Biochemical Studies - Carbohydrates *10068
Biochemical Studies - Minerals *10069
Movement *12100
Pathology, General and Miscellaneous - Therapy *12512
Routes of Immunization, Infection and Therapy *22100
BC Hominidae 86215
IT Major Concepts
Oncology (Human Medicine, Medical Sciences); Pharmacology
IT Parts, Structures, & Systems of Organisms
autologous hematopoietic progenitor cells: blood and lymphatics, cellular immunity reconstitution, transplantation, drug-induced mobilization, natural immunity reconstitution, humoral immunity reconstitution, high-dose chemotherapy support
IT Diseases
breast cancer: drug treatment, reproductive system disease/female, neoplastic disease, immunology
IT Chemicals & Biochemicals
carmustine: antineoplastic - drug, high-dose administration, combination therapy; cisplatin: antineoplastic - drug, combination therapy, high-dose administration; cyclophosphamide: antineoplastic - drug, combination therapy, high-dose administration; granulocyte colony-stimulating factor [Filgrastim]: hematologic - drug; stem cell factor [STEMGEN]: hematologic - drug
IT Miscellaneous Descriptors
Meeting Abstract; Meeting Poster
ORGN Super Taxa

Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae): female, patient
ORGN Organism Superterms
Animals; Chordates; Humans; Mammals; Primates; Vertebrates
RN 50-18-0 (CYCLOPHOSPHAMIDE)
15663-27-1 (CISPLATIN)
154-93-8 (CARMUSTINE)
121181-53-1 (FILGRASTIM)

L124 ANSWER 12 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:426598 BIOSIS
DN PREV199799725801
TI The effect of SCF, LIF, or Flt3L in combination with IL3 and IL6 on the retroviral gene transduction of hematopoietic stem cells.
AU Tushinski, R.; De Vries, P.; Moon, J.; Polikof, D.; Boehnlein, E.; Tsukamoto, A.
CS SyStemix Inc., Palo Alto, CA USA
SO Experimental Hematology (Charlottesville), (1997) Vol. 25, No. 8, pp. 890.
Meeting Info.: **26th Annual Meeting of the International Society for Experimental Hematology** Cannes, France August 24-28, 1997
ISSN: 0301-472X.
DT **Conference; Abstract**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines *10062
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Biochemical Studies - Carbohydrates *10068
Biophysics - Molecular Properties and Macromolecules *10506
Biophysics - Membrane Phenomena *10508
Enzymes - Chemical and Physical *10806
Enzymes - Physiological Studies *10808
Movement *12100
Pathology, General and Miscellaneous - Therapy *12512
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Metabolism - Nucleic Acids, Purines and Pyrimidines *13014
Digestive System - Physiology and Biochemistry *14004
Digestive System - Pathology *14006
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Reproductive System - Physiology and Biochemistry *16504
Reproductive System - Pathology *16506
Endocrine System - General *17002
Endocrine System - Thymus *17016
Neoplasms and Neoplastic Agents - Immunology *24003
Neoplasms and Neoplastic Agents - Biochemistry *24006
Neoplasms and Neoplastic Agents - Blood and Reticuloendothelial Neoplasms *24010
Developmental Biology - Embryology - General and Descriptive *25502
Developmental Biology - Embryology - Morphogenesis, General *25508
Genetics of Bacteria and Viruses *31500
Microbiological Apparatus, Methods and Media *32000
In Vitro Studies, Cellular and Subcellular *32600
Virology - Animal Host Viruses *33506
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508

Medical and Clinical Microbiology - Virology *36006
BC Retroviridae 02623
Hominidae *86215
IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Development; Digestive System (Ingestion and Assimilation); Endocrine System (Chemical Coordination and Homeostasis); Enzymology (Biochemistry and Molecular Biophysics); Gastroenterology (Human Medicine, Medical Sciences); Genetics; Hematology (Human Medicine, Medical Sciences); Infection; Membranes (Cell Biology); Metabolism; Methods and Techniques; Microbiology; Oncology (Human Medicine, Medical Sciences); Pathology; Physiology; Reproductive System (Reproduction)
IT Chemicals & Biochemicals
NEOMYCIN PHOSPHOTRANSFERASE; THYMIDINE KINASE
IT Miscellaneous Descriptors
BLOOD AND LYMPHATIC DISEASE; BLOOD AND LYMPHATICS; BREAST CANCER; CD34-POSITIVE CELL; CELL CULTURE; CELL DIFFERENTIATION; CULTURE METHOD; ENDOCRINE SYSTEM; FETAL LIVER KINASE-2 RECEPTOR LIGAND; FLT3L; GENE THERAPY; GENE THERAPY METHOD; GENE TRANSFER METHOD; HEMATOPOIETIC STEM CELLS; HIV REV PROTEIN; HUMAN IMMUNODEFICIENCY VIRUS REV PROTEIN; IL-3; IL-6; IMMUNE SYSTEM; IMMUNE SYSTEM DISEASE; INTERLEUKIN-3; INTERLEUKIN-6; LEUKEMIA INHIBITORY FACTOR; LIF; MICROBIOLOGICAL METHOD; MOBILIZED PERIPHERAL BLOOD; MOLECULAR GENETICS; MULTIPLE MYELOMA; MURINE LEUKEMIA VIRUS LONG TERMINAL REPEAT; NEOMYCIN PHOSPHOTRANSFERASE GENE; NEOPLASTIC DISEASE; REPRODUCTIVE SYSTEM DISEASE/FEMALE; RETROVIRAL GENE TRANSDUCTION; SCF; **STEM CELL FACTOR**; THYMIDINE KINASE PROMOTER; VECTOR
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia; Retroviridae: Viruses
ORGN Organism Name
human (Hominidae); retrovirus (Retroviridae)
ORGN Organism Superterms
animals; chordates; humans; mammals; microorganisms; primates; vertebrates; viruses
RN 62213-36-9 (NEOMYCIN PHOSPHOTRANSFERASE)
9002-06-6 (THYMIDINE KINASE)

L124 ANSWER 13 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:144506 BIOSIS
DN PREV199799443709
TI Site-specific requirements for interactions between c-Kit and its ligand during development and growth of TCR gamma-delta T cells.
AU Puddington, L. (1); Lewis, J.; Lefrancois, L. (1); Tigelaar, R.
CS (1) UCONN Health Cent., Farmington, CT USA
SO Journal of Allergy and Clinical Immunology, (1997) Vol. 99, No. 1 PART 2, pp. S242.
Meeting Info.: **Joint Meeting of the American Academy of Allergy, Asthma and Immunology, the American Association of Immunologists and the Clinical Immunology Society** San Francisco, California, USA February 21-26, 1997
ISSN: 0091-6749.
DT **Conference; Abstract**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Biophysics - Molecular Properties and Macromolecules *10506
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Integumentary System - Physiology and Biochemistry *18504
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae *86375

IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Cell Biology; Immune System (Chemical Coordination and Homeostasis); Integumentary System (Chemical Coordination and Homeostasis)
IT Miscellaneous Descriptors
ADULT; ALPHA BETA T CELL RECEPTOR; BLOOD AND LYMPHATICS; C-KIT STEM CELL FACTOR RECEPTOR; DENDRITIC EPIDERMAL T CELL; DIGESTIVE SYSTEM; GAMMA SIGMA T CELLS; IMMUNE SYSTEM; INTEGUMENTARY SYSTEM; INTESTINAL INTRAEPITHELIAL LYMPHOCYTES; NEWBORN; THYMUS
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates

L124 ANSWER 14 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:143939 BIOSIS
DN PREV199799443142
TI Induction of high affinity IgE receptor (Fc-epsilon-RI) on human mast cells by IL-4.
AU Toru, H. (1); Ra, C.; Nonoyama, S. (1); Suzuki, K.; Yata, J. (1); Nakahata, T.
CS (1) Tokyo Medical Dental Univ., Tokyo Japan
SO Journal of Allergy and Clinical Immunology, (1997) Vol. 99, No. 1 PART 2, pp. S103.
Meeting Info.: **Joint Meeting of the American Academy of Allergy, Asthma and Immunology, the American Association of Immunologists and the Clinical Immunology Society** San Francisco, California, USA February 21-26, 1997
ISSN: 0091-6749.
DT **Conference; Abstract**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Biophysics - Molecular Properties and Macromolecules *10506
Biophysics - Membrane Phenomena *10508
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Membranes (Cell Biology)
IT Miscellaneous Descriptors
ALPHA-CHAIN MESSENGER RNA; BIOCHEMISTRY AND BIOPHYSICS; FC-EPSILON-RI; HIGH AFFINITY IMMUNOGLOBULIN-E RECEPTOR; IMMUNE SYSTEM; IMMUNOGLOBULIN-E-MEDIATED ALLERGIC REACTION; INDUCTION; INTERLEUKIN-4; INTERLEUKIN-6; MAST CELLS; STEM CELL FACTOR
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms

animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 15 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:143906 BIOSIS
DN PREV199799443109
TI Murine embryonic yolk sac cells cultured with stem cell factor and interleukin-3 yield only unipotential mast cell colonies.
AU Desimone, S. K.; Klisch, G.; Huff, T. F.
CS Medical Coll. Virginia Campus/Virginia Commonwealth Univ., Richmond, VA USA
SO Journal of Allergy and Clinical Immunology, (1997) Vol. 99, No. 1 PART 2, pp. S95.
Meeting Info.: Joint Meeting of the American Academy of Allergy, Asthma and Immunology, the American Association of Immunologists and the Clinical Immunology Society San Francisco, California, USA February 21-26, 1997
ISSN: 0091-6749.
DT Conference; Abstract
LA English
CC General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520
Cytology and Cytochemistry - Animal *02506
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Reproductive System - Physiology and Biochemistry *16504
Endocrine System - General *17002
Developmental Biology - Embryology - General and Descriptive *25502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae *86375
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Development; Endocrine System (Chemical Coordination and Homeostasis); Immune System (Chemical Coordination and Homeostasis); Reproductive System (Reproduction)
IT Miscellaneous Descriptors
BLOOD AND LYMPHATICS; BONE MARROW; DEVELOPMENT; EMBRYO; EMBRYONIC STRUCTURE; EMBRYONIC YOLK SAC CELLS; IMMUNE SYSTEM; INTERLEUKIN-3; STEM CELL FACTOR; STRAIN-BALB/C; UNIPOTENTIAL MAST CELL COLONIES
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates

L124 ANSWER 16 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:143901 BIOSIS
DN PREV199799443104
TI Recombinant human (rh) GM-CSF, but not rhG-CSF, down-regulates the rhSCF-dependent differentiation of human fetal liver-derived mast cells.
AU Du, Z.; Li, Y.; Xia, H.-Z.; Irani, A. A.; Schwartz, L. B.
CS Virginia Commonwealth Univ., Richmond, VA USA
SO Journal of Allergy and Clinical Immunology, (1997) Vol. 99, No. 1 PART 2, pp. S94.
Meeting Info.: Joint Meeting of the American Academy of Allergy, Asthma and Immunology, the American Association of Immunologists and the Clinical Immunology Society San Francisco, California, USA February 21-26, 1997
ISSN: 0091-6749.
DT Conference; Abstract

LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Digestive System - Physiology and Biochemistry *14004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae *86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Digestive System (Ingestion and Assimilation); Endocrine System (Chemical Coordination and Homeostasis)
IT Miscellaneous Descriptors
BLOOD AND LYMPHATICS; DEPENDENT DIFFERENTIATION; FETUS; GROWTH FACTORS; IMMUNE SYSTEM; LIVER-DERIVED MAST CELLS; RECOMBINANT GRANULOCYTE-MACROPHAGE COLONY STIMULATING FACTOR; RECOMBINANT HUMAN **STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 17 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:54804 BIOSIS
DN PREV199799354007
TI Engraftment of cultured human hematopoietic cells in sheep.
AU Shimizu, Y. (1); Kobayashi, M.; Laver, J. H.; Almeida-Porada, G.; Zanjani, E. D.; Ogawa, M.
CS (1) Dep. Med., Med. Univ. South Carolina, Charleston, SC USA
SO Blood, (1996) Vol. 88, No. 10 SUPPL. 1 PART 1-2, pp. 456A.
Meeting Info.: **Thirty-eighth Annual Meeting of the American Society of Hematology** Orlando, Florida, USA December 6-10, 1996
ISSN: 0006-4971.
DT **Conference; Abstract**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Biochemical Studies - Carbohydrates *10068
Anatomy and Histology, General and Comparative - Regeneration and Transplantation *11107
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Reproductive System - Physiology and Biochemistry *16504
Reproductive System - Pathology *16506
Endocrine System - General *17002
Bones, Joints, Fasciae, Connective and Adipose Tissue - Physiology and Biochemistry *18004
Bones, Joints, Fasciae, Connective and Adipose Tissue - Pathology *18006

Developmental Biology - Embryology - Descriptive Teratology and Teratogenesis *25552
In Vitro Studies, Cellular and Subcellular *32600
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Bovidae 85715
Hominidae *86215
IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Development; Endocrine System (Chemical Coordination and Homeostasis); Hematology (Human Medicine, Medical Sciences); Metabolism; Physiology; Reproductive System (Reproduction); Skeletal System (Movement and Support)
IT Chemicals & Biochemicals
ERYTHROPOIETIN
IT Miscellaneous Descriptors
ADULT; ANALYTICAL METHOD; BLOOD AND LYMPHATICS; BONE MARROW; BONE MARROW TRANSPLANTATION; CD34-POSITIVE C-KIT-LOW CELLS; CD45-POSITIVE CELLS; CELL CULTURE; CELL EXPANSION; CELL PROCESSING; CELL SELECTION; ENGRAFTMENT; ERYTHROPOIETIN; FETUS; FLK2/FLT3 LIGAND; HEMATOPOIETIC CELLS; IL-3; IL-6; IN UTERO TRANSPLANTATION; INTERLEUKIN-3; INTERLEUKIN-6; **STEM CELL FACTOR**
ORGN Super Taxa
Bovidae: Artiodactyla, Mammalia, Vertebrata, Chordata, Animalia; Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae); sheep (Bovidae)
ORGN Organism Superterms
animals; artiodactyls; chordates; humans; mammals; nonhuman mammals; nonhuman vertebrates; primates; vertebrates
RN 11096-26-7 (ERYTHROPOIETIN)

L124 ANSWER 18 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:54530 BIOSIS
DN PREV199799353733
TI Superior mobilization of peripheral blood progenitor cells (PBPC) with r-metHuSCF (SCF) and r-metHuG-CSF (Filgrastim) in heavily pretreated multiple myeloma (MM) patients.
AU Tricot, G. (1); Jagnnath, S.; Desikan, K. R.; Siegel, D.; Munshi, N.; Olson, E.; Wyres, M.; Parker, W.; Barlogie, B.
CS (1) Univ. Arkansas Medical Sci., Little Rock, AR USA
SO Blood, (1996) Vol. 88, No. 10 SUPPL. 1 PART 1-2, pp. 388A.
Meeting Info.: **Thirty-eighth Annual Meeting of the American Society of Hematology** Orlando, Florida, USA December 6-10, 1996
ISSN: 0006-4971.
DT **Conference; Abstract; Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human 02508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Pathology, General and Miscellaneous - Therapy 12512
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Pharmacology - Clinical Pharmacology *22005
Pharmacology - Blood and Hematopoietic Agents *22008
Pharmacology - Immunological Processes and Allergy *22018
Toxicology - Pharmacological Toxicology *22504

Neoplasms and Neoplastic Agents - Immunology *24003
Neoplasms and Neoplastic Agents - Therapeutic Agents; Therapy *24008
Neoplasms and Neoplastic Agents - Blood and Reticuloendothelial Neoplasms *24010
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Hematology (Human Medicine, Medical Sciences); Oncology (Human Medicine, Medical Sciences); Pathology; Pharmacology; Toxicology
IT Chemicals & Biochemicals
FILGRASTIM; DIPHENHYDRAMINE
IT Miscellaneous Descriptors
ANTIHISTAMINE-DRUG; BLOOD AND LYMPHATIC DISEASE; BLOOD AND LYMPHATICS; DIPHENHYDRAMINE; FILGRASTIM; HEMATOLOGIC-DRUG; HEMATOLOGY; HYPERSENSITIVITY REACTION; IMMUNE SYSTEM DISEASE; MOBILIZATION; MULTIPLE MYELOMA; NEOPLASTIC DISEASE; ONCOLOGY; PATIENT; PERIPHERAL BLOOD PROGENITOR CELL; PHARMACOLOGY; R-METHUG-CSF; R-METHUSCF; RECOMBINANT HUMAN COLONY STIMULATING FACTOR; RECOMBINANT HUMAN **STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN 121181-53-1 (FILGRASTIM)
58-73-1 (DIPHENHYDRAMINE)

L124 ANSWER 19 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1997:53724 BIOSIS
DN PREV199799352927
TI Enhancement of donor (human) hematopoietic stem cell (HSC) engraftment in sheep co-transplanted in utero with human IL-3 producing stroma.
AU Almeida-Porada, G. D. (1); Nolta, J.; Tran, N.; Dao, M. A.; Zanjani, E. D.
CS (1) VAMC, Univ. Nevada, Reno, NV USA
SO Blood, (1996) Vol. 88, No. 10 SUPPL. 1 PART 1-2, pp. 186A.
Meeting Info.: **Thirty-eighth Annual Meeting of the American Society of Hematology** Orlando, Florida, USA December 6-10, 1996
ISSN: 0006-4971.
DT **Conference; Abstract; Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Biochemical Studies - Carbohydrates *10068
Anatomy and Histology, General and Comparative - Experimental Anatomy *11104
Anatomy and Histology, General and Comparative - Surgery *11105
Anatomy and Histology, General and Comparative - Regeneration and Transplantation *11107
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - General; Methods *15001
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004

Blood, Blood-Forming Organs and Body Fluids - Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Reproductive System - General; Methods *16501
Reproductive System - Physiology and Biochemistry *16504
Reproductive System - Pathology *16506
Endocrine System - General *17002
Bones, Joints, Fasciae, Connective and Adipose Tissue - General; Methods *18001
Bones, Joints, Fasciae, Connective and Adipose Tissue - Physiology and Biochemistry *18004
Bones, Joints, Fasciae, Connective and Adipose Tissue - Pathology *18006
Developmental Biology - Embryology - Experimental *25504
Developmental Biology - Embryology - Descriptive Teratology and Teratogenesis *25552
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508

BC Bovidae 85715
Hominidae *86215

IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Development; Endocrine System (Chemical Coordination and Homeostasis); Hematology (Human Medicine, Medical Sciences); Immune System (Chemical Coordination and Homeostasis); Metabolism; Morphology; Physiology; Reproductive System (Reproduction); Skeletal System (Movement and Support); Surgery (Medical Sciences)

IT Miscellaneous Descriptors
BLOOD AND LYMPHATICS; BONE MARROW CD34-POSITIVE CELLS; CYTOKINES; DONOR; DONOR HEMATOPOIETIC STEM CELL ENGRAFTMENT; FETUS; GM-CSF; GRANULOCYTE-MACROPHAGE COLONY-STIMULATING FACTOR; HEMATOPOIETIC MICROENVIRONMENT; HEMATOPOIETIC STEM CELL; HUMAN IL-3; HUMAN IL-3 PRODUCING STROMA; HUMAN IL-3 PRODUCING STROMA TRANSPLANTATION; HUMAN INTERLEUKIN-3; HUMAN/SHEEP XENOGRAFT; IL-6; IN-UTERO COTRANSPLANTATION; INTERLEUKIN-6; METHODOLOGY; PRODUCTION; RECIPIENT; SCF; **STEM CELL FACTOR**; STROMAL CELLS; SURGICAL METHOD

ORGN Super Taxa
Bovidae: Artiodactyla, Mammalia, Vertebrata, Chordata, Animalia; Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name
human (Hominidae); sheep (Bovidae)

ORGN Organism Superterms
animals; artiodactyls; chordates; humans; mammals; nonhuman mammals; nonhuman vertebrates; primates; vertebrates

L124 ANSWER 20 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:550616 BIOSIS
DN PREV199699272972
TI Characterization of keratinocyte- and fibroblast-derived mitogens for human melanocytes: Their roles in stimulated cutaneous pigmentation.
AU Imokawa, Genji (1); Yada, Yukihiro; Morisaki, Naoko; Kimura, Mitsutoshi
CS (1) Inst. Fundamental Res., Kao Corporation, 2602 Akabane, Ichikai-Machi, Haga, Tochigi 321-34 Japan
SO Hori, Y. [Editor]; Hearing, V. J. [Editor]; Nakayama, J. [Editor]. **International Congress Series**, (1996) No. 1096, pp. 35-48.
International Congress Series; Melanogenesis and malignant melanoma: Biochemistry, cell biology, molecular biology, pathophysiology, diagnosis and treatment.
Publisher: Elsevier Science Publishers B.V. PO Box 211, Sara Burgerhartstraat 25, 1000 AE Amsterdam, Netherlands.
Meeting Info.: **International Symposium on Melanogenesis and Malignant Melanoma** Fukuoka, Japan December 4-6, 1995
ISSN: 0531-5131. ISBN: 0-444-82209-7.
DT Book; **Conference**

LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human 02508
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Metabolism - Nucleic Acids, Purines and Pyrimidines *13014
Endocrine System - General *17002
Integumentary System - Physiology and Biochemistry *18504
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae *86215
IT Major Concepts
Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Integumentary System (Chemical Coordination and Homeostasis); Metabolism
IT Miscellaneous Descriptors
BOOK CHAPTER; DNA SYNTHESIS; ENDOTHELIN-1; EPIDERMAL MELANOSIS; GRANULOCYTE-MACROPHAGE COLONY STIMULATING FACTOR; INTERLEUKIN-1 ALPHA; **MEETING** PAPER; **STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 21 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:450913 BIOSIS
DN PREV199699173269
TI Amplification of the lymphoid compartments in serum-derived cultures of human cord blood cells.
AU Sanchez, M. (1); Pascuccio, M.; Barca, A.; Migliaccio, A. R.; Migliaccio, G.
CS (1) Ist. Superiore Sanita, Rome Italy
SO Experimental Hematology (Charlottesville), (1996) Vol. 24, No. 9, pp. 1099.
Meeting Info.: **25th Annual Meeting of the International Society for Experimental Hematology** New York, New York, USA August 23-27, 1996
ISSN: 0301-472X.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Reproductive System - Physiology and Biochemistry *16504
Endocrine System - General *17002
Developmental Biology - Embryology - Morphogenesis, General *25508
Tissue Culture, Apparatus, Methods and Media *32500
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae *86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Development; Endocrine System (Chemical Coordination and Homeostasis); Methods and Techniques; Reproductive System (Reproduction)
IT Miscellaneous Descriptors
BLOOD AND LYMPHATICS; CORD BLOOD CELLS; CYTOKINE; INTERLEUKIN-2; INTERLEUKIN-4; INTERLEUKIN-7; LYMPHOID COMPARTMENT AMPLIFICATION; **MEETING ABSTRACT; STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name

human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 22 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:450573 BIOSIS
DN PREV199699172929
TI Ontogeny-related predominance of multipotent lymphomyeloid stem cells in human fetal liver.
AU Zijlmans, J. M. J. M.; Duinkerken, N.; Lim, F. T. H.; Melenhorst, J. J.; Willemze, R.; Fibbe, W. E.
CS Lab. Exp. Hematol., Dep. Hematol., Univ. Med. Cent., Leiden Netherlands
SO Experimental Hematology (Charlottesville), (1996) Vol. 24, No. 9, pp. 1035.
Meeting Info.: **25th Annual Meeting of the International Society for Experimental Hematology** New York, New York, USA August 23-27, 1996
ISSN: 0301-472X.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Digestive System - Physiology and Biochemistry *14004
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Developmental Biology - Embryology - Morphogenesis, General *25508
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae *86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Development; Digestive System (Ingestion and Assimilation); Endocrine System (Chemical Coordination and Homeostasis); Genetics
IT Miscellaneous Descriptors
B LYMPHOCYTE; CELL DIFFERENTIATION; HEMATOPOIESIS; HLA; INTERLEUKIN-4; INTERLEUKIN-7; **MEETING ABSTRACT**; POLYMERASE CHAIN REACTION; **STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 23 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:300563 BIOSIS
DN PREV199699022919
TI Evidence that **stem cell factor** (SCF) modulates neuroimmune interactions: Mast cell (MC) activation by substance P is influenced by SCF via A G protein-dependent mechanism.
AU Furuta, G. T. (1); Williams, R. E.; Lavigne, J. A.; Galli, S. J.; Wershil, B. K.
CS (1) Combined Program Pediatric GI/Nutrition, Harvard Med. Sch., Boston, MA USA
SO Gastroenterology, (1996) Vol. 110, No. 4 SUPPL., pp. A911.
Meeting Info.: **96th Annual Meeting of the American Gastroenterological Association and the Digestive Disease Week** San Francisco, California, USA May 19-22, 1996
ISSN: 0016-5085.

DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Digestive System - Pathology *14006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Endocrine System - Neuroendocrinology *17020
Nervous System - Physiology and Biochemistry *20504
In Vitro Studies, Cellular and Subcellular *32600
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae *86375
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Digestive System (Ingestion and Assimilation); Endocrine System (Chemical Coordination and Homeostasis); Immune System (Chemical Coordination and Homeostasis); Nervous System (Neural Coordination); Pathology
IT Chemicals & Biochemicals
SUBSTANCE P; NEUROKININ A; NEUROKININ B
IT Miscellaneous Descriptors
BONE MARROW CELL; **MEETING ABSTRACT**; NEUROKININ A; NEUROKININ B; SUBSTANCE P
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates
RN 33507-63-0 (SUBSTANCE P)
86933-74-6 (NEUROKININ A)
102577-23-1 (NEUROKININ B)

L124 ANSWER 24 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:145013 BIOSIS
DN PREV199698717148
TI Immunolocalization of **stem cell factors** in inflamed human nasal tissues.
AU Kim, Y. K.; Nakagawa, N.; Sulakvelidze, I.; Dolovich, J.; Denburg, J. A.
CS Hamilton, ON Canada
SO Journal of Allergy and Clinical Immunology, (1996) Vol. 97, No. 1 PART 3, pp. 282.
Meeting Info.: **Fifty-second Annual Meeting of the American Academy of Allergy Asthma and Immunology** New Orleans, Louisiana, USA March 15-20, 1996
ISSN: 0091-6749.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Social Biology; Human Ecology *05500
Ecology; Environmental Biology - Animal *07508
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Pathology, General and Miscellaneous - Comparative *12503
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008

Respiratory System - Physiology and Biochemistry *16004
Respiratory System - Pathology *16006
Endocrine System - General *17002
Toxicology - General; Methods and Experimental *22501
Developmental Biology - Embryology - Morphogenesis, General *25508
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Development; Ecology (Environmental Sciences); Endocrine System (Chemical Coordination and Homeostasis); Human Ecology (Anthropology); Pathology; Pulmonary Medicine (Human Medicine, Medical Sciences); Respiratory System (Respiration); Toxicology
IT Miscellaneous Descriptors
ALLERGIC INFLAMMATORY DISEASE; DIFFERENTIATION; EPITHELIAL CELL; IMMUNOLOGY; MAST CELL PROGENITOR; **MEETING ABSTRACT**; NASAL POLYP; PROLIFERATION; SEASONAL RHINITIS
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 25 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:145012 BIOSIS
DN PREV199698717147
TI Regulation of production of **stem cell factor** (SCF) by fibroblasts: Role of serum factors.
AU Finotto, S.; Horowitz, J.; McLaren, R.; Busse, P. J.; Metcalfe, D. D.
CS Bethesda, MD USA
SO Journal of Allergy and Clinical Immunology, (1996) Vol. 97, No. 1 PART 3, pp. 281.
Meeting Info.: **Fifty-second Annual Meeting of the American Academy of Allergy Asthma and Immunology** New Orleans, Louisiana, USA March 15-20, 1996
ISSN: 0091-6749.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines *10062
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Blood, Blood-Forming Organs and Body Fluids - Blood and Lymph Studies *15002
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Integumentary System - Physiology and Biochemistry *18504
Developmental Biology - Embryology - Morphogenesis, General *25508
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Development; Endocrine System (Chemical Coordination and Homeostasis); Integumentary System (Chemical Coordination and

Homeostasis)
IT Miscellaneous Descriptors
ALLERGY; HEMATOPOIETIC GROWTH FACTOR; IMMUNOLOGY; ISOFORM; MAST CELL GROWTH FACTOR; **MEETING ABSTRACT**; MESSENGER RNA; SKIN
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 26 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:144931 BIOSIS
DN PREV199698717066
TI Histamine release from human skin mast cells by monocyte chemoattractant factor: 1. RANTES, macrophage inflammatory protein - 1-alpha, and **stem cell factor** using microdialysis technique.
AU Petersen, L. J.; Brasso, K.; Pryds, M.; Skov, P. S.
CS Copenhagen Denmark
SO Journal of Allergy and Clinical Immunology, (1996) Vol. 97, No. 1 PART 3, pp. 261.
Meeting Info.: **Fifty-second Annual Meeting of the American Academy of Allergy Asthma and Immunology** New Orleans, Louisiana, USA March 15-20, 1996
ISSN: 0091-6749.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Cytology and Cytochemistry - Human *02508
Clinical Biochemistry; General Methods and Applications *10006
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Bones, Joints, Fasciae, Connective and Adipose Tissue - Physiology and Biochemistry *18004
Integumentary System - Physiology and Biochemistry *18504
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae 86215
Muridae *86375
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Chemistry (Allied Medical Sciences); Clinical Immunology (Human Medicine, Medical Sciences); Integumentary System (Chemical Coordination and Homeostasis); Metabolism; Skeletal System (Movement and Support)
IT Chemicals & Biochemicals
HISTAMINE
IT Miscellaneous Descriptors
BASOPHILS; CHEMOKINES; **MEETING ABSTRACT**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia; Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae); rat (Muridae); Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; nonhuman mammals; nonhuman vertebrates; primates; rodents; vertebrates
RN 51-45-6 (HISTAMINE)

L124 ANSWER 27 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1996:48637 BIOSIS
DN PREV199698620772
TI Comparison of retroviral transduction conditions for gene marking of adult peripheral blood or marrow-derived CD34+ cells in a clinical trial.
AU Emmons, R. V. B. (1); Doren, S.; Hines, K.; Carter, C. S.; Cottler-Fox, M.; O'Shaughnessy, J. A.; Leitman, S. F.; Cowan, K.; Dunbar, C. E.
CS (1) Hematology Branch, NHLBI, Med. Branch, NCI, Bethesda, MD USA
SO Blood, (1995) Vol. 86, No. 10 SUPPL. 1, pp. 238A.
Meeting Info.: **37th Annual Meeting of the American Society of Hematology** Seattle, Washington, USA December 1-5, 1995
ISSN: 0006-4971.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Anatomy and Histology, General and Comparative - Regeneration and Transplantation *11107
Pathology, General and Miscellaneous - Therapy *12512
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Reproductive System - Pathology *16506
Endocrine System - General *17002
Pharmacology - Blood and Hematopoietic Agents *22008
Pharmacology - Reproductive System; Implantation Studies *22028
Neoplasms and Neoplastic Agents - Therapeutic Agents; Therapy *24008
Neoplasms and Neoplastic Agents - Blood and Reticuloendothelial Neoplasms *24010
Genetics of Bacteria and Viruses *31500
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Retroviridae 02623
Hominidae *86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Genetics; Hematology (Human Medicine, Medical Sciences); Oncology (Human Medicine, Medical Sciences); Pathology; Pharmacology; Physiology; Reproductive System (Reproduction)
IT Miscellaneous Descriptors
AUTOLOGOUS TRANSPLANTATION; BREAST CANCER; HIGH-DOSE CHEMOTHERAPY; INTERLEUKIN-3; INTERLEUKIN-6; **MEETING ABSTRACT**; **MEETING POSTER**; MULTIPLE MYELOMA; **STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia; Retroviridae: Viruses
ORGN Organism Name
human (Hominidae); Retroviridae (Retroviridae)
ORGN Organism Superterms
animals; chordates; humans; mammals; microorganisms; primates; vertebrates; viruses

L124 ANSWER 28 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:518577 BIOSIS
DN PREV199598532877
TI In lethally irradiated mice interleukin-12 protects bone marrow but sensitizes intestinal tract to damage from ionizing radiation.
AU Neta, R.; Stiefel, S. M.; Ali, N.

CS Dep. Experimental Hematol., Armed Forces Radiobiol. Res. Inst., Bethesda, MD 20889-5603 USA
SO Mackiewicz, A. [Editor]; Koj, A. [Editor]; Sehgal, P. B. [Editor]. Annals of the New York Academy of Sciences, (1995) Vol. 762, pp. 274-281. Annals of the New York Academy of Sciences; Interleukin-6-type cytokines. Publisher: New York Academy of Sciences 2 East 63rd Street, New York, New York 10021, USA.
Meeting Info.: **Conference** Poznan, Poland June 19-22, 1994
ISSN: 0077-8923. ISBN: 0-89766-932-0 (paper), 0-89766-931-2 (cloth).
DT Book; **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal 02506
Radiation - Radiation Effects and Protective Measures *06506
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biophysics - General Biophysical Techniques 10504
Digestive System - Pathology *14006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae *86375
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Digestive System (Ingestion and Assimilation); Endocrine System (Chemical Coordination and Homeostasis); Immune System (Chemical Coordination and Homeostasis); Radiation Biology
IT Miscellaneous Descriptors
BOOK CHAPTER; **MEETING** PAPER; RADIOPROTECTION; **STEM CELL FACTOR**
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Muridae (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman vertebrates; nonhuman mammals; rodents; vertebrates

L124 ANSWER 29 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:423965 BIOSIS
DN PREV199598438265
TI A feasibility study of ex vivo expansion of CD34 positive peripheral blood progenitor cells (PBPC) on a clinical scale.
AU Holyoake, T. L. (1); Alcorn, M. J. (1); Richmond, L. (1); Pearson, C. (1); Farrell, E. (1); Kyle, B.; Dunlop, D. J. (1); Fitzsimons, E.; Pragnell, I. B.; Franklin, I. M. (1)
CS (1) Glasgow Royal Infirmary, Glasgow UK
SO Experimental Hematology (Charlottesville), (1995) Vol. 23, No. 8, pp. 760.
Meeting Info.: **24th Annual Meeting of the International Society for Experimental Hematology** Duesseldorf, Germany August 27-31, 1995
ISSN: 0301-472X.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Anatomy and Histology, General and Comparative - Regeneration and Transplantation *11107
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008

Reproductive System - Pathology *16506
Endocrine System - General *17002
Neoplasms and Neoplastic Agents - Therapeutic Agents; Therapy *24008
Neoplasms and Neoplastic Agents - Blood and Reticuloendothelial Neoplasms *24010
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae *86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Hematology (Human Medicine, Medical Sciences); Oncology (Human Medicine, Medical Sciences); Physiology; Reproductive System (Reproduction)
IT Chemicals & Biochemicals
ERYTHROPOIETIN
IT Miscellaneous Descriptors
BREAST CANCER; ERYTHROPOIETIN; INTERLEUKINS; LYMPHOMA; **MEETING ABSTRACT; MEETING POSTER;** MULTIPLE MYELOMA; MYELOSUPPRESSION; **STEM CELL FACTOR;** TRANSPLANTATION
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN 11096-26-7 (ERYTHROPOIETIN)

L124 ANSWER 30 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:421590 BIOSIS
DN PREV199598435890
TI Human keratinocytes release mast cell differentiation factors other than **stem cell factor.**
AU Welker, Pia (1); Grabbe, Juergen; Czarnetzki, Beate M.
CS (1) Freie Univ. Berlin, Rudolf Virchow Clin. Dermatol., Augustenburger-Platz 1, D-13344 Berlin Germany
SO International Archives of Allergy and Immunology, (1995) Vol. 107, No. 1-3, pp. 139-141.
Meeting Info.: **20th Symposium of the Collegium Internationale Allergologicum on Molecular and Clinical Implications for Allergy in the 21st Century** Nantucket, Massachusetts, USA September 25-29, 1994
ISSN: 1018-2438.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Enzymes - Physiological Studies *10808
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies 15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Bones, Joints, Fasciae, Connective and Adipose Tissue - Anatomy *18002
Bones, Joints, Fasciae, Connective and Adipose Tissue - Physiology and Biochemistry *18004
Integumentary System - Anatomy *18502
Integumentary System - Physiology and Biochemistry *18504
Developmental Biology - Embryology - Morphogenesis, General *25508

Tissue Culture, Apparatus, Methods and Media 32500
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae *86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Development; Enzymology (Biochemistry and Molecular Biophysics); Genetics; Immune System (Chemical Coordination and Homeostasis); Integumentary System (Chemical Coordination and Homeostasis); Metabolism; Pathology; Skeletal System (Movement and Support)
IT Chemicals & Biochemicals
HISTAMINE; TRYPTASE
IT Miscellaneous Descriptors
FIBROBLAST; HISTAMINE; HUMAN HACAT KERATINOCYTE CELL LINE; HUMAN HMC-1 MAST CELL LINE; **MEETING ABSTRACT; MEETING** PAPER; TRYPTASE
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN 51-45-6 (HISTAMINE)
97501-93-4 (TRYPTASE)

L124 ANSWER 31 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:421562 BIOSIS
DN PREV199598435862
TI Regulation of mouse and human mast cell development, survival and function by **stem cell factor**, the ligand for the c-kit receptor.
AU Galli, Stephen J. (1); Tsai, Mindy; Wershil, Barry K.; Tam, See-Ying; Costa, John J.
CS (1) Dep. Pathol., Beth Isr. Hosp., 330 Brookline Ave., Boston, MA 02215 USA
SO International Archives of Allergy and Immunology, (1995) Vol. 107, No. 1-3, pp. 51-53.
Meeting Info.: **20th Symposium of the Collegium Internationale Allergologicum on Molecular and Clinical Implications for Allergy in the 21st Century** Nantucket, Massachusetts, USA September 25-29, 1994
ISSN: 1018-2438.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Cytology and Cytochemistry - Human *02508
Genetics and Cytogenetics - Animal *03506
Genetics and Cytogenetics - Human *03508
Biochemical Methods - Proteins, Peptides and Amino Acids *10054
Biochemical Methods - Carbohydrates *10058
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Replication, Transcription, Translation *10300
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Pathology, General and Miscellaneous - Necrosis *12510
Metabolism - Carbohydrates *13004
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies 15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Bones, Joints, Fasciae, Connective and Adipose Tissue - Anatomy *18002

Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae 86215
Muridae *86375
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Genetics; Metabolism; Methods and Techniques; Molecular Genetics (Biochemistry and Molecular Biophysics); Pathology; Skeletal System (Movement and Support)
IT Miscellaneous Descriptors
ALLERGY; ANAPHYLAXIS; APOPTOSIS; BCL-2; C-KIT; IMMUNOGLOBULIN E ANTIBODY; MAST CELL-DEFICIENT MOUSE; MASTOCYTOSIS; **MEETING ABSTRACT**; **MEETING** PAPER; PATHOGENESIS; PROTO-ONCOGENES
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia; Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae); Muridae (Muridae)
ORGN Organism Superterms
animals; chordates; humans; mammals; nonhuman mammals; nonhuman vertebrates; primates; rodents; vertebrates

L124 ANSWER 32 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:380847 BIOSIS
DN PREV199598395147
TI The role of **stem cell factor** (C-kit ligand) and inflammatory cytokines in pulmonary mast cell activation.
AU Lukacs, N. W. (1); Kunkel, S. L. (1); Evanoff, H. (1); Strieter, R. M.; Key, M. L.; Kunkel, R. G. (1); Taub, D. D.
CS (1) Univ. Michigan Med. Sch., Dep. Pathol., NCI-FCRDC, Fredrick, MD USA
SO 9TH INTERNATIONAL **CONGRESS** OF IMMUNOLOGY.. (1995) pp. 40. The 9th International **Congress** of Immunology.
Publisher: 9th International **Congress** of Immunology San Francisco, California, USA.
Meeting Info.: **Meeting Sponsored by the American Association of Immunologists and the International Union of Immunological Societies** San Francisco, California, USA July 23-29, 1995
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Respiratory System - Physiology and Biochemistry *16004
Respiratory System - Pathology *16006
Endocrine System - General *17002
Developmental Biology - Embryology - Morphogenesis, General *25508
In Vitro Studies, Cellular and Subcellular *32600
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Muridae *86375
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Development; Endocrine System (Chemical Coordination and Homeostasis); Immune System (Chemical Coordination and Homeostasis); Pathology; Respiratory System (Respiration)
IT Miscellaneous Descriptors

ALLERGIC AIRWAY RESPONSE; INTERFERON-ALPHA; INTERLEUKIN-10; INTERLEUKIN-3; INTERLEUKIN-4; MAST CELL PROLIFERATION; **MEETING ABSTRACT**
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates

L124 ANSWER 33 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:197839 BIOSIS
DN PREV199598212139
TI Identification and immunotyping of committed non-granulated mast cell precursors in the peripheral blood of a patient with aggressive systemic mastocytosis.
AU Castells, M. (1); Friend, D.; Bunnell, C.; Austen, K. F.
CS (1) Brigham and Women's Hosp., Boston, MA 02115 USA
SO FASEB Journal, (1995) Vol. 9, No. 4, pp. A1047.
Meeting Info.: Experimental Biology 95, Part II Atlanta, Georgia, USA April 9-13, 1995
ISSN: 0892-6638.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Blood, Blood-Forming Organs and Body Fluids - Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Blood and Lymphatics (Transport and Circulation); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Hematology (Human Medicine, Medical Sciences); Immune System (Chemical Coordination and Homeostasis)
IT Miscellaneous Descriptors
BONE MARROW; DIFFERENTIATION; MATURATION; **MEETING ABSTRACT; STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 34 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:196426 BIOSIS
DN PREV199598210726
TI Role of c-kit ligand in allergic airway eosinophilia.
AU Lukacs, N. W. (1); Strieter, R. M.; Lincoln, P.; Kunkel, S. L.
CS (1) Univ. Mich. Med. Sch., Dep. Pathol., Div. Pulmonary Critical Care Med., Ann Arbor, MI USA
SO FASEB Journal, (1995) Vol. 9, No. 4, pp. A803.
Meeting Info.: Experimental Biology 95, Part II Atlanta, Georgia, USA April 9-13, 1995

ISSN: 0892-6638.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human 02508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Respiratory System - Pathology *16006
Endocrine System - General *17002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Pathology; Pulmonary Medicine (Human Medicine, Medical Sciences)
IT Miscellaneous Descriptors
INFLAMMATION; **MEETING ABSTRACT**; PULMONARY DISEASE; **STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 35 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:194729 BIOSIS
DN PREV199598209029
TI Pulmonary mast cell-derived chemokines.
AU Kunkel, R. G. (1); Strieter, R. M.; Kunkel, S. L. (1); Evanoff, H. L. (1); Lukacs, N. W. (1)
CS (1) Univ. Mich. Med. Sch., Dep. Pathol., Ann Arbor, MI USA
SO FASEB Journal, (1995) Vol. 9, No. 3, pp. A511.
Meeting Info.: Experimental Biology 95, Part I Atlanta, Georgia, USA April 9-13, 1995
ISSN: 0892-6638.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Respiratory System - Physiology and Biochemistry *16004
Endocrine System - General *17002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Muridae *86375
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Immune System (Chemical Coordination and Homeostasis); Pathology; Respiratory System

(Respiration)
IT Miscellaneous Descriptors
ALLERGEN SPECIFIC DEGRANULATION; INFLAMMATION; INTERLEUKIN-3; **MEETING ABSTRACT; STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia; Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae); Muridae (Muridae)
ORGN Organism Superterms
animals; chordates; humans; mammals; nonhuman mammals; nonhuman vertebrates; primates; rodents; vertebrates

L124 ANSWER 36 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:190180 BIOSIS
DN PREV199598204480
TI Interactions between c-kit and **stem cell factor** are required for intestinal immune system homeostasis.
AU Puddington, Lynn; Olson, Sara; Lefrancois, Leo
CS Univ. Conn. Health Center, Farmington, CT 06030 USA
SO Journal of Cellular Biochemistry Supplement, (1995) Vol. 0, No. 19A, pp. 250.
Meeting Info.: **Keystone Symposium on Mucosal Immunity: New Strategies for Protection Against Viral and Bacterial Pathogens** Keystone, Colorado, USA January 16-23, 1995
ISSN: 0733-1959.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Genetics and Cytogenetics - Animal *03506
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Replication, Transcription, Translation *10300
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Metabolism - Proteins, Peptides and Amino Acids *13012
Digestive System - Pathology *14006
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae *86375
IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Cell Biology; Digestive System (Ingestion and Assimilation); Genetics; Immune System (Chemical Coordination and Homeostasis); Metabolism; Molecular Genetics (Biochemistry and Molecular Biophysics); Pathology
IT Miscellaneous Descriptors
CD4-POSITIVE; CD8-POSITIVE; DEVELOPMENT; INTRAEPITHELIAL LYMPHOCYTES; **MEETING ABSTRACT; MEETING POSTER;** MUCOSAL IMMUNE RESPONSE
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mice (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates

L124 ANSWER 37 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:143356 BIOSIS

DN PREV199598157656
TI Human nasal polyp fibroblasts produce **stem cell factor** (SCF.
AU Nakagawa, N.; Howie, K.; Switzer, J.; Marshall, J.; Denburg, J. A.
CS Hamilton, ON Canada
SO Journal of Allergy and Clinical Immunology, (1995) Vol. 95, No. 1 PART 2, pp. 292.
Meeting Info.: **Fifty-first Annual Meeting of the American Academy of Allergy and Immunology** New York, New York, USA February 24-March 1, 1995
ISSN: 0091-6749.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System 15008
Respiratory System - Pathology *16006
Endocrine System - General *17002
Bones, Joints, Fasciae, Connective and Adipose Tissue - Physiology and Biochemistry *18004
Tissue Culture, Apparatus, Methods and Media 32500
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Genetics; Metabolism; Pathology; Pulmonary Medicine (Human Medicine, Medical Sciences); Skeletal System (Movement and Support)
IT Miscellaneous Descriptors
ALLERGIC AIRWAY DISEASE; C-KIT GENE EXPRESSION; INFLAMMATION; MAST CELL ACTIVATION; **MEETING ABSTRACT**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 38 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1995:47052 BIOSIS
DN PREV199598061352
TI Effect of c-kit ligand on human intestinal mast cells.
AU Schwengberg, S. (1); Bischoff, S. C. (1); Wordelmann, K. (1); Raab, H. R.; Dralle, H.; Meyer, H. J.; Manns, M. P. (1)
CS (1) Dep. Gastroenterol., Hannover Med. Sch., Hannover Germany
SO Immunobiology, (1994) Vol. 191, No. 2-3, pp. 190.
Meeting Info.: **XXVth Meeting of the Society of Immunology** Konstanz, Germany September 21-24, 1994
ISSN: 0171-2985.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Anatomy and Histology, General and Comparative - Regeneration and

Transplantation *11107
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Digestive System - Physiology and Biochemistry *14004
Endocrine System - General *17002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Digestive System (Ingestion and Assimilation); Endocrine System (Chemical Coordination and Homeostasis); Pathology; Physiology
IT Miscellaneous Descriptors
ALLERGIC REACTION; INFLAMMATION; **MEETING ABSTRACT**; REGENERATION; **STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 39 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1994:328471 BIOSIS
DN PREV199497341471
TI Topical tretinoin increases dermal mast cells and induces **stem cell factor** in hairless mice.
AU Kligman, Lorraine H.; Murphy, George F.
CS Dep. Dermatol., Univ. Pennsylvania, Philadelphia, PA USA
SO Journal of Investigative Dermatology, (1994) Vol. 102, No. 4, pp. 612.
Meeting Info.: **Annual Meeting of the Society for Investigative Dermatology** Baltimore, Maryland, USA April 27-30, 1994
ISSN: 0022-202X.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Biochemical Studies - Vitamins 10063
Biochemical Studies - Lipids 10066
Pathology, General and Miscellaneous - Therapy *12512
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Blood, Lymphatic and Reticuloendothelial Pathologies *15006
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Integumentary System - General; Methods *18501
Integumentary System - Pathology *18506
Pharmacology - Clinical Pharmacology 22005
Pharmacology - Integumentary System, Dental and Oral Biology *22020
Routes of Immunization, Infection and Therapy *22100
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae *86375
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Immune System (Chemical Coordination and Homeostasis); Integumentary System (Chemical Coordination and Homeostasis); Methods and Techniques; Pathology; Pharmacology
IT Chemicals & Biochemicals
TRETINOIN
IT Miscellaneous Descriptors
DERMATOLOGICAL-DRUG; **MEETING ABSTRACT**;

MEETING POSTER; PHARMACODYNAMICS; TRETINOIN
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Muridae (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman vertebrates; nonhuman mammals; rodents; vertebrates
RN 302-79-4 (TRETINOIN)

L124 ANSWER 40 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1994:150493 BIOSIS
DN PREV199497163493
TI Recombinant human **stem cell factor** (rhSCF) induces cutaneous mast cell activation and hyperplasia, and hyperpigmentation in humans in vivo.
AU Costa, J. J.; Demetri, G. D.; Harrist, T. J.; Dvorak, A. M.; Hayes, D. F.; Merica, E. A.; Menchaca, D. M.; Gringeri, A. J.; Galli, S. J.
CS Boston, MA USA
SO Journal of Allergy and Clinical Immunology, (1994) Vol. 93, No. 1 PART 2, pp. 225.
Meeting Info.: **Fiftieth Annual Meeting of the American Academy of Allergy and Immunology** Anaheim, California, USA March 4-9, 1994
ISSN: 0091-6749.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human *02508
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Endocrine System - General *17002
Bones, Joints, Fasciae, Connective and Adipose Tissue - General; Methods *18001
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Diptera 75314
Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Biochemistry and Molecular Biophysics; Cell Biology; Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Pathology; Skeletal System (Movement and Support)
IT Miscellaneous Descriptors
ALLERGY; **MEETING ABSTRACT**
ORGN Super Taxa
Diptera: Insecta, Arthropoda, Invertebrata, Animalia; Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae); Diptera (Diptera)
ORGN Organism Superterms
animals; arthropods; chordates; humans; insects; invertebrates; mammals; primates; vertebrates

L124 ANSWER 41 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1994:93764 BIOSIS
DN PREV199497106764
TI A novel mutation affecting the second immunoglobulin-like domain of the human kit receptor.
AU Fleischman, R. A.
CS Markey Cancer Cent. and VA Hosp., Univ. Kentucky Med. Cent., Lexington, KY USA
SO Blood, (1993) Vol. 82, No. 10 SUPPL. 1, pp. 231A.

Meeting Info.: **Thirty-fifth Annual Meeting of the American Society of Hematology** St. Louis, Missouri, USA December 3-7, 1993
ISSN: 0006-4971.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biophysics - Molecular Properties and Macromolecules *10506
Biophysics - Membrane Phenomena *10508
Enzymes - Physiological Studies *10808
Metabolism - Proteins, Peptides and Amino Acids *13012
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Integumentary System - Pathology *18506
Developmental Biology - Embryology - Morphogenesis, General *25508
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae *86215
IT Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Dermatology (Human Medicine, Medical Sciences); Development; Endocrine System (Chemical Coordination and Homeostasis); Enzymology (Biochemistry and Molecular Biophysics); Genetics; Membranes (Cell Biology); Metabolism
IT Chemicals & Biochemicals
TYROSINE KINASE
IT Miscellaneous Descriptors
ADHESION; HEMATOPOIETIC GROWTH FACTOR; LIGAND BINDING; **MEETING ABSTRACT**; **MEETING POSTER**; PIEBALDISM; RECEPTOR DIMERIZATION; **STEM CELL FACTOR** BINDING; TYROSINE KINASE
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
human (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN 80449-02-1 (TYROSINE KINASE)

L124 ANSWER 42 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1993:334146 BIOSIS
DN PREV199345028871
TI Effects of chronic treatment with the c-kit ligand, **stem cell factor**, on IgE-dependent anaphylaxis in mice: Genetically mast cell-deficient Sl/Sl-d mice acquire anaphylactic responsiveness, but the congenic normal mice do not exhibit augmented responses.
AU Ando, A. (1); Martin, T. R.; Galli, S. J.
CS (1) Dep. Pathol., Beth Israel Hosp., Harvard Med. Sch., Boston, MA 02215 USA
SO Journal of Immunology, (1993) Vol. 150, No. 8 PART 2, pp. 178A.
Meeting Info.: **Joint Meeting of the American Association of Immunologists and the Clinical Immunology Society** Denver, Colorado, USA May 21-25, 1993
ISSN: 0022-1767.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506

Genetics and Cytogenetics - Human *03508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Pharmacology - Immunological Processes and Allergy *22018
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Muridae *86375
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Cell Biology; Genetics; Immune System (Chemical Coordination and Homeostasis); Pharmacology
IT Miscellaneous Descriptors
ABSTRACT; IMMUNOGLOBULIN E; IMMUNOLOGIC-DRUG
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Muridae (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman vertebrates; nonhuman mammals; rodents; vertebrates

L124 ANSWER 43 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1993:201090 BIOSIS
DN PREV199344097340
TI Modulation of human lung mast cell function by the c-kit receptor ligand.
AU De Paulis, Amato; Ciccarelli, Anna; Cirillo, Raffaele; De Crescenzo, Gennaro; Columbo, Michele; Marone, Gianni
CS Cattedra Immunol. Clin. Allergol., II Fac. Med. Chirurgia, Univ. Napoli Federico II, Via S. Pansini 5, I-80131 Naples Italy
SO International Archives of Allergy and Immunology, (1992) Vol. 99, No. 2-4, pp. 326-329.
Meeting Info.: **19th CIA (Collegium Internationale Allergologicum) Symposium on Chemical Mediators and Cellular Interactions in Clinical Immunology** Capri, Italy May 3-7, 1992
ISSN: 1018-2438.
DT Article
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human 02508
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Lipids 10066
Biophysics - Membrane Phenomena 10508
Enzymes - Physiological Studies *10808
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Respiratory System - Physiology and Biochemistry *16004
Endocrine System - General *17002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae *86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Enzymology (Biochemistry and Molecular Biophysics); Genetics; Respiratory System (Respiration)
IT Chemicals & Biochemicals
TYROSINE KINASE; HISTAMINE; PROSTAGLANDIN D2; LEUKOTRIENE C4
IT Miscellaneous Descriptors
HISTAMINE; LEUKOTRIENE C4; PROSTAGLANDIN D2; PROTO-ONCOGENE; **STEM CELL FACTOR**; TYROSINE KINASE
ORGN Super Taxa

Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN 80449-02-1 (TYROSINE KINASE)
51-45-6 (HISTAMINE)
41598-07-6 (PROSTAGLANDIN D2)
72025-60-6 (LEUKOTRIENE C4)

L124 ANSWER 44 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1993:201089 BIOSIS
DN PREV199344097339
TI Effect of recombinant human c-kit receptor ligand on mediator release from human skin mast cells.
AU Columbo, Michele (1); Horowitz, Edward M.; Botana, Luis M.; Macglashan., Donald W., Jr.; Bochner, Bruce S.; Gillis, Steven; Zsebo, Krisztina M.; Galli, Stephen J.; Lichtensen, Lawrence M.
CS (1) Div. Immunol. Clin., Ist. Med. Interna Cardiol. Chirurgia Cardiovasc., II Fac. Med. Chirurgia, Univ. Napoli Federico II, Via S. Pansini 5, I-80131 Naples Italy
SO International Archives of Allergy and Immunology, (1992) Vol. 99, No. 2-4, pp. 323-325.
Meeting Info.: **19th CIA (Collegium Internationale Allergologicum) Symposium on Chemical Mediators and Cellular Interactions in Clinical Immunology** Capri, Italy May 3-7, 1992
ISSN: 1018-2438.
DT Article
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human 02508
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Lipids 10066
Biochemical Studies - Minerals 10069
Metabolism - Minerals *13010
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Integumentary System - Physiology and Biochemistry *18504
In Vitro Studies, Cellular and Subcellular 32600
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae *86215
IT Major Concepts
Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Genetics; Integumentary System (Chemical Coordination and Homeostasis); Metabolism
IT Chemicals & Biochemicals
CALCIUM; HISTAMINE
IT Miscellaneous Descriptors
CALCIUM; HISTAMINE; IMMUNOGLOBULIN E; PROSTAGLANDIN D- 2; **STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates
RN 7440-70-2 (CALCIUM)
51-45-6 (HISTAMINE)

L124 ANSWER 45 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1993:179863 BIOSIS

DN PREV199344087463
TI Expression of IL-4 mRNA in human dermal mast cells in response to Fc receptor crosslinkage in the presence of SCF.
AU Okayama, Y. (1); Quint, D.; Hunt, T. C. (1); El-Lati, S. (1); Heusser, C. H.; Bullock, G.; Mueller, R.; Bradding, P. (1); Howarth, P. (1); et al.
CS (1) Immunopharmacol. Group, University Southampton UK
SO Journal of Allergy and Clinical Immunology, (1993) Vol. 91, No. 1 PART 2, pp. 256.
Meeting Info.: **Forty-ninth Annual Meeting of the American Academy of Allergy and Immunology** Chicago, Illinois, USA March 12-17, 1993
ISSN: 0091-6749.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Carbohydrates 10068
Replication, Transcription, Translation *10300
Pathology, General and Miscellaneous - Inflammation and Inflammatory Disease *12508
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Integumentary System - Pathology *18506
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
Allergy *35500
BC Hominidae *86215
IT Major Concepts
Allergy (Clinical Immunology, Human Medicine, Medical Sciences); Blood and Lymphatics (Transport and Circulation); Clinical Immunology (Human Medicine, Medical Sciences); Dermatology (Human Medicine, Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Genetics; Molecular Genetics (Biochemistry and Molecular Biophysics); Pathology
IT Miscellaneous Descriptors
ABSTRACT; ALLERGY; INFLAMMATION; INTERLEUKIN-4 MESSENGER RNA; PATHOGENESIS; **STEM CELL FACTOR**
ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
Hominidae (Hominidae)
ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L124 ANSWER 46 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1993:87198 BIOSIS
DN PREV199344041448
TI Antigenic heterogeneity and tumor progression in cutaneous malignant melanoma (CMM.
AU Natali, P. G.; Nicotra, M. R.; Cavaliere, F.; Bigotti, A.
CS Regina Elena Cancer Inst., Rome Italy
SO Anticancer Research, (1992) Vol. 12, No. 6A, pp. 1846.
Meeting Info.: **Fourth International Conference of Anticancer Research** Rethymnon, Crete, Greece October 21-25, 1992
ISSN: 0250-7005.
DT **Conference**
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Genetics and Cytogenetics - Animal *03506
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biophysics - Molecular Properties and Macromolecules *10506

Biophysics - Membrane Phenomena *10508
Integumentary System - Physiology and Biochemistry *18504
Integumentary System - Pathology *18506
Neoplasms and Neoplastic Agents - Diagnostic Methods *24001
Neoplasms and Neoplastic Agents - Pathology; Clinical Aspects; Systemic Effects *24004
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae *86375
IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Genetics; Immune System (Chemical Coordination and Homeostasis); Integumentary System (Chemical Coordination and Homeostasis); Membranes (Cell Biology); Tumor Biology
IT Chemicals & Biochemicals
INTEGRIN
IT Miscellaneous Descriptors
ABSTRACT; C-KIT RECEPTOR-**STEM CELL FACTOR** COMPLEX; CYTOKINE PRODUCTION; IMMUNOPATHOLOGY; INTEGRIN PHENOTYPE; INTERCELLULAR ADHESION MOLECULE-1; LAMININ; TENASCIN
ORGN Super Taxa
Muridae: Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGN Organism Name
mouse (Muridae)
ORGN Organism Superterms
animals; chordates; mammals; nonhuman mammals; nonhuman vertebrates; rodents; vertebrates
RN 153-87-7Q (INTEGRIN)
60791-49-3Q (INTEGRIN)

L124 ANSWER 47 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1992:378921 BIOSIS
DN BR43:45871
TI **STEM CELL FACTOR** MAINTAINS THE VIABILITY AND FUNCTION OF CULTURED RAT PERITONEAL MAST CELLS.
AU WU S V; WEI J Y; HONG L S; WANG Y H; GO V L W
CS DEP. MED., BRI AND CURE/DDC, UCLA, LOS ANGELES, CALIF.
SO DIGESTIVE DISEASE WEEK AND THE 93RD ANNUAL **MEETING** OF THE AMERICAN GASTROENTEROLOGICAL ASSOCIATION, SAN FRANCISCO, CALIFORNIA, USA, MAY 9-15, 1992. GASTROENTEROLOGY. (1992) 102 (4 PART 2), A766.
CODEN: GASTAB. ISSN: 0016-5085.
DT **Conference**
FS BR; OLD
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines *10062
Digestive System - Anatomy *14002
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae 86375
IT Miscellaneous Descriptors
ABSTRACT DNA SYNTHESIS

L124 ANSWER 48 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1992:378703 BIOSIS
DN BR43:45653
TI BONE MARROW-DERIVED CULTURED MAST CELLS BMCMC GROWN IN **STEM CELL FACTOR** MATURE AND ACQUIRE RESPONSIVENESS TO SUBSTANCE P SP WHICH INDUCES THE CELLS TO RELEASE HISTAMINE AND TUMOR NECROSIS FACTOR-ALPHA TNF-ALPHA.
AU WERSHIL B K; LAVIGNE J A; ZSEBO K M; GALLI S J
CS DEP. PATHOL., BETH ISR. HOSP., BOSTON, MASS., USA.
SO DIGESTIVE DISEASE WEEK AND THE 93RD ANNUAL **MEETING** OF THE

AMERICAN GASTROENTEROLOGICAL ASSOCIATION, SAN FRANCISCO, CALIFORNIA, USA, MAY 9-15, 1992. GASTROENTEROLOGY. (1992) 102 (4 PART 2), A712.
CODEN: GASTAB. ISSN: 0016-5085.

DT **Conference**
FS BR; OLD
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Animal *02506
Biochemical Studies - Proteins, Peptides and Amino Acids *10064
Digestive System - Pathology *14006
Blood, Blood-Forming Organs and Body Fluids - Blood Cell Studies *15004
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Bones, Joints, Fasciae, Connective and Adipose Tissue - Anatomy *18002
Nervous System - Physiology and Biochemistry *20504
Immunology and Immunochemistry - General; Methods *34502
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Muridae 86375
IT Miscellaneous Descriptors
ABSTRACT MOUSE CYTOKINE
RN 51-45-6 (HISTAMINE)
33507-63-0 (SUBSTANCE P)

L124 ANSWER 49 OF 49 BIOSIS COPYRIGHT 2000 BIOSIS
AN 1992:203218 BIOSIS
DN BR42:96293
TI RECOMBINANT HUMAN **STEM CELL FACTOR** RHSCF IS AN ACTIVATOR-MODULATOR OF MEDIATOR RELEASE FROM HUMAN SKIN MAST CELLS.
AU COLUMBO M; HOROWITZ E M; BOTANA L M; MACGLASHAN D W JR; ZSEBO K M; GALL S J; LICHTENSTEIN L M
CS JOHNS HOPKINS MED. SCH., BALTIMORE, MD.
SO FORTY-EIGHTH ANNUAL **MEETING** OF THE AMERICAN ACADEMY OF ALLERGY AND IMMUNOLOGY, ORLANDO, FLORIDA, USA, MARCH 6-11, 1992. J ALLERGY CLIN IMMUNOL. (1992) 89 (1 PART 2), 243.
CODEN: JACIBY. ISSN: 0091-6749.

DT **Conference**
FS BR; OLD
LA English
CC **General Biology - Symposia, Transactions and Proceedings of Conferences, Congresses, Review Annuals 00520**
Cytology and Cytochemistry - Human 02508
Biochemical Studies - Proteins, Peptides and Amino Acids 10064
Biochemical Studies - Lipids 10066
Biochemical Studies - Minerals 10069
Metabolism - Minerals 13010
Metabolism - Proteins, Peptides and Amino Acids 13012
Blood, Blood-Forming Organs and Body Fluids - Lymphatic Tissue and Reticuloendothelial System *15008
Endocrine System - General *17002
Integumentary System - Physiology and Biochemistry *18504
Immunology and Immunochemistry - Immunopathology, Tissue Immunology *34508
BC Hominidae 86215
IT Miscellaneous Descriptors
ABSTRACT IMMUNOGLOBULIN E SUBSTANCE P PROSTAGLANDIN D-2 CALCIUM
RN 7440-70-2 (CALCIUM)
33507-63-0 (SUBSTANCE P)
41598-07-6 (PROSTAGLANDIN D-2)

=> fil wpids

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=> d his l127-


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     FILE 'WPIDS' ENTERED AT 10:33:48 ON 28 JUN 2000
L127        685 S STEM CELL FACTOR
L128         23 S STEM CELL FACTOR/TI
L129          5 S L128 AND (PREVENT? OR ANTIBOD?)/TI
L130         46 S L127 AND (SIGNAL? OR TRANSDUC?)
L131          0 S L127 AND ACK2
L132          1 S L130 AND CLINICAL/TI
L133          6 S L129,L132

     FILE 'WPIDS' ENTERED AT 10:43:24 ON 28 JUN 2000

=> d all abeq tech tot


L133 ANSWER 1 OF 6  WPIDS COPYRIGHT 2000   DERWENT INFORMATION LTD
AN   1999-508554 [42]   WPIDS
CR   2000-293134 [25]
DNC  C1999-148555
TI   Controlling the proliferation and differentiation of stem cells or
     progenitor cells, used in clinical applications.
DC   B04 D16
IN   FIBACH, E; FRIEDMAN, M M; PELED, T; TREVES, A
PA   (GAMI-N) GAMIDA CELL LTD; (HADA-N) HADASIT MEDICAL RES SERVICES & DEV
CYC  84
PI   WO 9940783    A1 19990819 (199942)* EN   60p    A01N001-02
        RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL
            OA PT SD SE SZ UG ZW
         W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD
            GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV
            MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT
            UA UG US UZ VN YU ZW
     AU 9926624    A  19990830 (200003)              A01N001-02
ADT  WO 9940783 A1 WO 1999-US2664 19990208; AU 9926624 A AU 1999-26624 19990208
FDT  AU 9926624 A Based on WO 9940783
PRAI US 1998-130367   19980807; US 1998-24195    19980217
IC   ICM  A01N001-02
     ICS  A61K035-12; C12N015-85; C12N015-86; C12P019-34
AB   WO   9940783 A UPAB: 20000524
     NOVELTY - Expanding a population of cells, while at the same time
     inhibiting differentiation of the cells by providing the cells with
     conditions for cell proliferation and, at the same time, for reducing a
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capacity of the cells in utilizing transition metals, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) hemopoietic cells transplantation comprising:

(a) obtaining hemopoietic cells to be transplanted from a donor;

(b) providing the cells ex vivo with conditions for cell proliferation and, at the same time, for reducing a capacity of the cells in utilizing transition metals to expand a population of the cells, while at the same time, inhibiting differentiation of the cells; and

(c) transplanting the cells to a patient;

(2) **transducing** stem cells with an exogene comprising:

(a) obtaining stem cells to be **transduced**;

(b) as in (1b), and

(c) **transducing** the cells with the exogene;

(3) adoptive immuno-therapy comprising:

(a) obtaining progenitor hematopoietic cells from a patient, and

(b) as in (1b) and (1c);

(4) mobilization of bone marrow stem cells into the peripheral blood of a donor for harvesting the cells comprising:

(a) administering to the donor an agent for reducing a capacity of the cells in utilizing transition metals, to expand a population of stem cells, while at the same time, inhibiting differentiation of the stem cells; and

(b) harvesting the cells by leukapheresis;

(5) decelerating maturation/differentiation of erythroid precursor cells for the treatment of beta -hemoglobinopathic patients comprising administering to the patient an agent as in (4a), such that upon natural removal of the agent from the body, the stem cells undergo accelerated maturation resulting in elevated production of fetal hemoglobin;

(6) a therapeutical ex vivo cultured cell preparation comprising ex vivo cells propagated in presence of an agent as in (4a);

(7) preservation of stem cells comprising handling the stem cell in at least one of the steps selected from harvesting, isolation and storage, in a presence of a transition metal chelator, and

(8) stem cells collection bags, separation and washing buffers supplemented with an effective amount or concentration of a transition metal chelator, which inhibits cell differentiation.

USE - The expansion of stem cells and other defined lympho-hemopoietic cell subpopulations by ex-vivo culturing is especially useful in clinical applications.

ADVANTAGE - In order to achieve maximal ex vivo expansion of stem cells the following conditions should be fulfilled:

(i) differentiation should be reversibly inhibited or delayed, and

(ii) self-renewal should be maximally prolonged.

The new methods satisfy these requirements.

Dwg.0/17

FS CPI

FA AB; DCN

MC CPI: B04-F02; B04-H02C; B04-H02G; B04-H04A; B05-A03A; B07-D11; B10-B01B; B14-H01; D05-H08; D05-H14B2

TECH UPTX: 19991014

TECHNOLOGY FOCUS - BIOTECHNOLOGY - Preferred Methods: The cells are in vivo, and the conditions for cell proliferation are naturally provided, whereas reducing the capacity of the cells in utilizing transition metals is effected by administering a transition metal chelator. Reducing the capacity of the cells in utilizing transition metals is further effected by administering zinc. Alternatively, the cells are ex vivo. Then providing the cells with the conditions for cell proliferation, include providing the cells with nutrients and with cytokines. The **transduction** in (2) is effected by a vector including the exogene. Preferred Cytokines: The cytokines are early acting cytokines selected from **stem cell factor**, FLT3 ligand, interleukin-6, thrombopoletin and interleukin-3. Alternatively, the cytokines are late acting cytokines selected from granulocyte colony stimulating factor, granulocyte/macrophage colony stimulating factor and erythropoietin.

Preferred Cells: The cells are selected from hematopoietic cells, neural cells and oligodendrocyte cells, skin cells, hepatic cells, muscle cells, bone cells, mesenchymal cells, pancreatic cells, chondrocytes and stroma cells. The cells are derived from a source selected from bone marrow, peripheral blood and neonatal umbilical cord blood. The cells are enriched for hematopoietic CD34+ cells. The cells are selected from non-differentiated stem cells and committed progenitor cells. The donor and the patient are a single individual.

TECHNOLOGY FOCUS - ORGANIC CHEMISTRY - Preferred Chelator: The transition metal chelator is selected from polyamine chelating agents, ethylendiamine, diethylenetriamine, triethylenetetramine, triethylenediamine, tetraethylenepentamine (TEPA), aminoethylethanolamine, aminoethylpiperazine, pentaethylenehexamine, triethylenetetramine-hydrochloride, tetraethylenepentamine-hydrochloride, pentaethylenehexamine-hydrochloride, tetraethylpentamine, captopril, penicilamine and transition metal binding peptides.

L133 ANSWER 2 OF 6 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1999-084645 [08] WPIDS
DNN N1999-061085 DNC C1999-025651
TI New monoclonal **antibody** specific for bovine derived **stem cell factor** - useful for producing hybridoma and method of bovine derived **stem cell factor** detection.
DC B04 D16 S03
PA (NORQ) NORINSUISANSHO KACHIKU EISEI; (NORI-N) ZH NORIN SUISAN SENTAN GIJUTSU SANGYO
CYC 1
PI JP 10313860 A 19981202 (199908)* JA 12p C12N015-02
ADT JP 10313860 A JP 1997-131437 19970521
PRAI JP 1997-131437 19970521
IC ICM C12N015-02
ICS C07K016-24; C12P021-08; G01N033-53; G01N033-577
ICI C12P021-08, C12R001:91
AB JP 10313860 A UPAB: 19990302
New monoclonal antibody specifically reacts to bovine-derived stem cell factor (SCF). Also claimed is a hybridoma producing the above monoclonal antibody.
USE - The antibody is useful in a method for determining bovine-derived SCF (claimed).
Dwg.0/8
FS CPI EPI
FA AB
MC CPI: B04-F05; B04-G21; D05-H08; D05-H11A
EPI: S03-E14H4

L133 ANSWER 3 OF 6 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1997-367060 [34] WPIDS
DNC C1997-117736
TI Monoclonal **antibody** to human **stem cell factor** - comprises specifically binding to human **stem cell factor**, useful in diagnosis of blood diseases..
DC B04 D16
PA (NCHK) NICHIREI KK
CYC 1
PI JP 09154578 A 19970617 (199734)* 5p C12N015-02
ADT JP 09154578 A JP 1995-335685 19951201
PRAI JP 1995-335685 19951201
IC ICM C12N015-02
ICS C07K016-18; C12N005-10; C12P021-08
ICI C12N005-10, C12R001:91; C12P021-08, C12R001:
AB JP 09154578 A UPAB: 19970820
A monoclonal antibody specifically binding to human stem cell factor is new.
Also claimed are: a hybridoma capable of producing the monoclonal

antibody; and a process for producing the monoclonal antibody by culturing the hybridoma.

The monoclonal antibody can prevent human stem cell factor from binding to a product of a human c-kit gene. The subclass of the monoclonal antibody is IgG.

USE - Human stem cell factor (SCF) is ligand of c-kit receptor expressed on haematopoietic stem cells and plays an important role in haematopoiesis, and it is of importance for diagnosis of various diseases including blood diseases. The monoclonal antibody of the invention binds specifically to SCF to diagnose such diseases. The monoclonal antibody can further be used as a therapeutic agent for such diseases.

ADVANTAGE - SCF is present in blood in 2 forms i.e. soluble type active SCF (free SCF) and membrane-bound inactive SCF (c-kit bound SCF). Because the monoclonal antibody binds to free SCF but not to c-kit-bound SCF, the antibody can be used to measure only the active SCF or to monitor artificially administered soluble type SCF, as opposed to a conventional measurement method where SCF is detected without distinguishing the 2 forms.

Dwg.0/2

FS CPI
FA AB
MC CPI: B04-F05; B04-G02; B04-G21; B12-K04A2; B14-F03; D05-H11A1; D05-H15

L133 ANSWER 4 OF 6 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1997-066617 [07] WPIDS
DNN N1997-054751 DNC C1997-021981
TI Monoclonal **antibody** to human **stem cell factor** receptor - for diagnostic and therapeutic use.
DC B04 D16 S03
IN BUEHRING, H
PA (UYTU-N) UNIV TUEBINGEN EBERHARD-KARLS
CYC 12
PI DE 19600589 C1 19970116 (199707)* 9p C07K016-22
EP 787743 A2 19970806 (199736) DE 10p C07K016-28
R: AT BE CH DE ES FR GB IT LI NL SE
EP 787743 A3 19970820 (199745) C07K016-22
US 5808002 A 19980915 (199844) C07K016-28
ADT DE 19600589 C1 DE 1996-19600589 19960110; EP 787743 A2 EP 1996-118320 19961115; EP 787743 A3 EP 1996-118320 19961115; US 5808002 A US 1997-778524 19970103
PRAI DE 1996-19600589 19960110
REP No-SR.Pub; 2.Jnl.Ref; WO 9217505; WO 9221766
IC ICM C07K016-22; C07K016-28
ICS A61K039-395; C12N005-16; C12N005-18; C12N005-20; C12P021-08; G01N033-53; G01N033-577
AB DE 19600589 C UPAB: 19970212
New monoclonal antibody that binds specifically to human stem cell factor (SCF) receptor is produced by hybridoma cell line A3C6E2 (DSM ACC 2247).

Also claimed are hybridoma cells that produce the antibody.

USE - The antibody is used in compsns. to treat tumours. The antibody can also be used to detect and isolate haematopoietic stem cells, e.g. for gene therapy treatment by retroviral-mediated gene transfer into the haematopoietic cells. The antibody can also be used to inhibit haematopoiesis. The antibodies can be used to inhibit the binding of SCF. The antibody can be used to modify the sensitivity of patients to cell-cycle-specific chemotherapeutic agents (all claimed).

Dwg.0/3

FS CPI EPI
FA AB
MC CPI: B04-F01; B04-F05; B04-G21; B04-K01; B11-C07A7; B12-K04A1; B14-H01A; D05-H11A1; D05-H15
EPI: S03-E14H4

L133 ANSWER 5 OF 6 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1993-207031 [26] WPIDS
DNC C1993-091695

TI **Stem cell factor** binding proteins e.g. Kit-X
- useful for treating and **preventing** SCF-associated diseases e.g. neoplasias, anaemia(s), myeloid leukaemia and glioblastoma.
DC B04 D16
IN GIVOL, D; YARDEN, Y
PA (YEDA) YEDA RES & DEV CO LTD
CYC 8
PI EP 548867 A2 19930630 (199326)* EN 24p C12N015-12
R: CH DE ES FR GB IT LI NL
EP 548867 A3 19940413 (199522) C12N015-12
ADT EP 548867 A2 EP 1992-121681 19921221; EP 548867 A3 EP 1992-121681 19921221
PRAI IL 1991-100469 19911223; IL 1992-103434 19921015
REP No-SR.Pub; 3.Jnl.Ref; WO 9010013; WO 9217505
IC ICM C12N015-12
ICS A61K037-02; C07K013-00; C12N005-10; C12P021-08
AB EP 548867 A UPAB: 19931116
The protein is of (a) a soluble SCF-receptor comprising the extra cellular domain of an SCF-receptor and (b) analogues of (a) obtd. by deletion, addn. or replacement of aminoacid residue(s) without affecting the binding properties to SCF.
Pref. the protein may be a soluble SCF-receptor designated Kit-X.
Pref. a conjugate comprises the protein bound to another bioactive molecule of cytoactive drugs, cytotoxins and antibodies.
USE - The protein is to SCF and acts as an antagonist for SCF-mediated bioactivities in the treatment and prevention of diseases e.g. hyperproliferative or neoplastic states of myeloid neuronal or germ cells, macrocytic anaemia, neutropenia, myeloid leukaemia, mastocytoma, glioblastoma and myelodysplasia. The protein may also be used for the detection, isolation and purificn. of SCF. Antibodies to the protein may be used for purifying the protien in assays for determining bioactivities of the protein and for detection of SCF-receptor.
Dwg.0/18
FS CPI
FA AB
MC CPI: B04-B02B1; B04-B04A1; B04-B04A3; B04-B04A6; B04-B04C6; B12-G01; B12-G05; B12-G07; B12-K04A; D05-C12; D05-H08; D05-H09; D05-H12

L133 ANSWER 6 OF 6 WPIDS COPYRIGHT 2000 DERWENT INFORMATION LTD
AN 1992-366198 [44] WPIDS
DNC C1992-162617
TI Monoclonal **antibodies** against **stem cell factor** receptors - for treating leukaemia(s) and solid tumours and for purifying haematopoietic cells.
DC B04 D16
IN BROUDY, V C; LIN, N
PA (UNIW) UNIV WASHINGTON; (AMGE-N) AMGEN INC
CYC 18
PI WO 9217505 A1 19921015 (199244)* EN 59p C07K015-28
RW: AT BE CH DE DK ES FR GB GR IT LU MC NL SE
W: CA JP
EP 578774 A1 19940119 (199403) EN C07K015-28
R: AT BE CH DE DK ES FR GB GR IT LI LU MC NL SE
JP 06506833 W 19940804 (199435) 18p C12P021-08
US 5489516 A 19960206 (199612) 17p G01N033-53
EP 578774 A4 19951115 (199626) C07K015-28
EP 578774 B1 19980729 (199834) EN C07K016-18
R: AT BE CH DE DK ES FR GB GR IT LI LU MC NL SE
DE 69226431 E 19980903 (199841) C07K016-18
ES 2118820 T3 19981001 (199848) C07K016-18
US 5906938 A 19990525 (199928) C12N015-85
US 5919911 A 19990706 (199933) A61K039-395
US 5922847 A 19990713 (199934) A61K035-14
ADT WO 9217505 A1 WO 1992-US2674 19920403; EP 578774 A1 EP 1992-910836 19920403, WO 1992-US2674 19920403; JP 06506833 W JP 1992-510017 19920403, WO 1992-US2674 19920403; US 5489516 A Cont of US 1991-681245 19910405, US 1993-11078 19930129; EP 578774 A4 EP 1992-910836 ; EP 578774 B1 EP

1992-910836 19920403, WO 1992-US2674 19920403; DE 69226431 E DE 1992-626431 19920403, EP 1992-910836 19920403, WO 1992-US2674 19920403; ES 2118820 T3 EP 1992-910836 19920403; US 5906938 A Cont of US 1991-681245 19910405, Cont of US 1993-11078 19930129, US 1995-449139 19950524; US 5919911 A Cont of US 1991-681245 19910405, Cont of US 1993-11078 19930129, US 1995-462638 19950605; US 5922847 A Cont of US 1991-681245 19910405, Div ex US 1993-11078 19930129, US 1994-255193 19940607

FDT EP 578774 A1 Based on WO 9217505; JP 06506833 W Based on WO 9217505; EP 578774 B1 Based on WO 9217505; DE 69226431 E Based on EP 578774, Based on WO 9217505; ES 2118820 T3 Based on EP 578774; US 5906938 A Cont of US 5489516; US 5919911 A Cont of US 5489516; US 5922847 A Div ex US 5489516

PRAI US 1991-681245 19910405; US 1993-11078 19930129; US 1995-449139 19950524; US 1995-462638 19950605; US 1994-255193 19940607

REP 10Jnl.Ref; No-Citns.

IC ICM A61K035-14; A61K039-395; C07K015-28; C07K016-18; C12N015-85; C12P021-08; G01N033-53

ICS A01N001-02; A61K039-44; C07K001-22; C07K016-28; C07K016-30; C12N005-18; C12N005-20; C12N015-02

AB WO 9217505 A UPAB: 19931116

A monoclonal antibody (MAb) having an ability to bind to a stem cell factor (SCF) receptor is claimed. Also claimed is a hybridoma capable of producing the MAb.

USE/ADVANTAGE - The MAb can be used for purifying haematopoietic cells. The purified cells can be used in bone marrow transplantation or gene therapy after retrovirally-mediated gene transfer into the purified cells. The MAb can also be used for sepg. normal cells from neoplastic cells based on differential numbers of SCF receptors. The MAb can also be conjugated.

Dwg.0/7

FS CPI

FA AB

MC CPI: B04-B04A3; B04-B04C5; B12-D02B; B12-G05; B12-G07; B12-K04A1; D05-H11

ABEQ US 5489516 A UPAB: 19960322

A monoclonal antibody produced by hybridoma cell line ATCC No. HB 10716. receptoris used against stem cell factor receptors for treating leukaemia and solid tumours and for purifying haematopoietic cells.

Dwg.0/7